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In this work the author has endeavored to meet certain demands, the urgency of which has been borne in upon his mind after more than thirty years' experience in the treatment of oral diseases and malformations. As a teacher of oral surgery he has also become convinced that there is great need of a text-book adapted to both didatic and recitational instruction, sufficiently concise and well systematized to allow a teacher to accommodate his course to the college curriculum and yet be able to impress the salient points to the best possible advantage. While meeting these requirements, it is believed that this work is sufficiently comprehensive and practical to make it a useful guide in trying clinical situations after graduation. The chapters on "Harelip" and "Cleft Palate" represent the chief part of the author's life-work. His presentation of these subjects is based upon extended observation of the confusing conditions which surround those who are called upon to treat such cases in the course of general practice. The clinical aspect has been kept constantly in view. Many original drawings have been carefully prepared to show each operative step in the correction of these deformities. The pictures of large numbers of patients are shown in order to emphasize the thoroughly practical value of the operations and corrective measures recommended.

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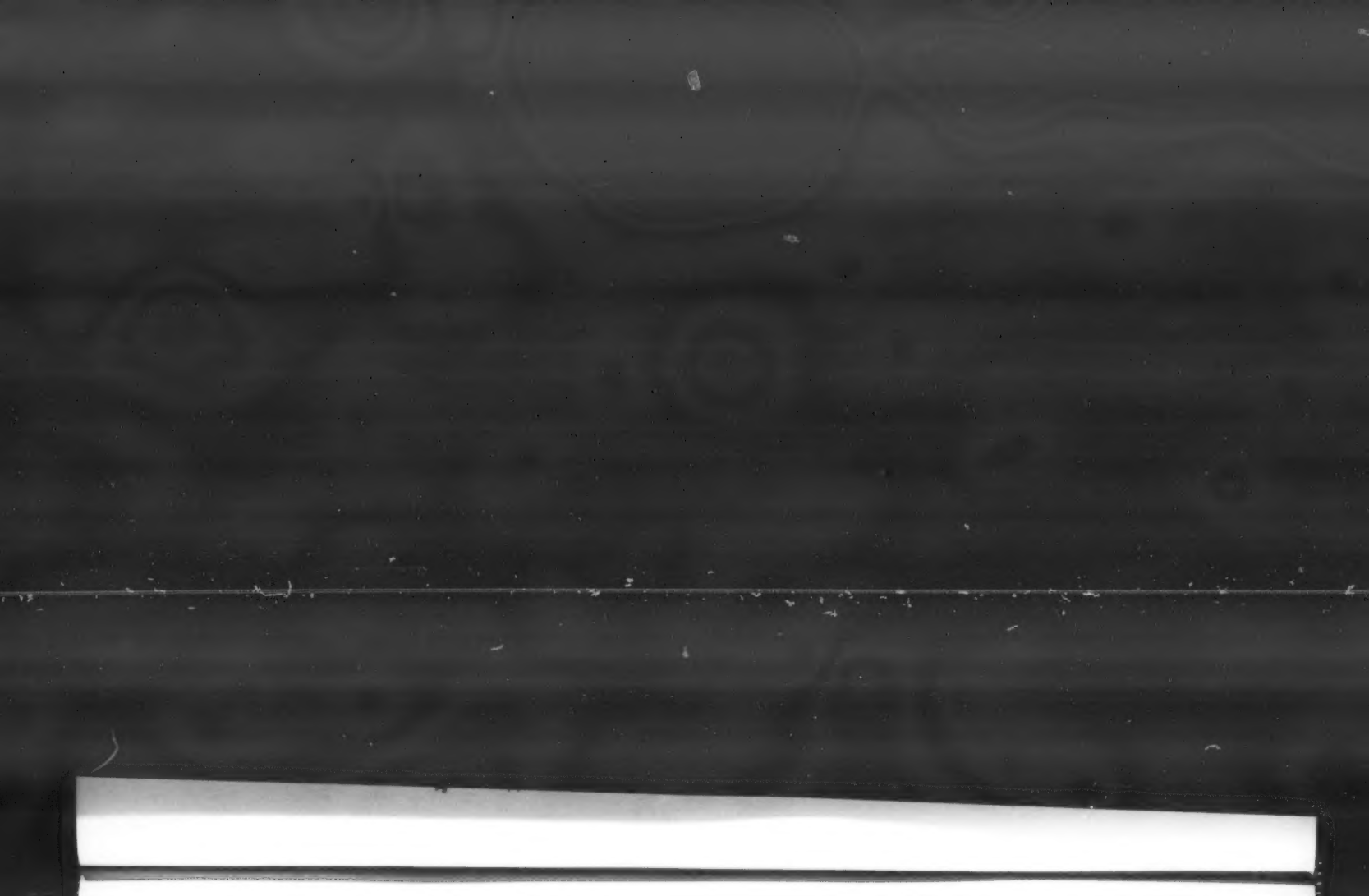

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THE

AMERICAN JOURNAL
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APRIL, 1912

ORIGINAL ARTICLES

JEJUNOSTOMY.

By William J. Mayo, M.D.,

SURGEON TO ST. MARY'S HOSPITAL, ROCHESTER, MINNESOTA.

Jejunostomy is an operation of considerable usefulness; it gives rest to the stomach and maintains good nutrition. The operation is an active competitor of gastrostomy in cases of esophageal and cardiac obstruction, and in extensive ulcers which cannot be excised and gastrojejunostomy is not feasible, it is a most valuable procedure. In malignant diseases of the stomach of the ulcerous type it offers a means of palliation.

The technique of jejunostomy is simple and easily carried out. The abdomen is opened by an epigastric incision either in the midline or to the left in the rectus muscle. The jejunum is picked up and, selecting a point from twelve to sixteen inches from its origin, a loop is drawn out of the abdomen, nicked on the convex surface (Fig. 1) and a No. 9 (English scale) rubber catheter pushed through the opening down stream until it extends about three inches inside the lumen of the jejunum (Fig. 2). This point is fixed in position by a single chromic catgut suture, the catheter is then infolded by the jejunal wall for an inch or an inch and a half by mattress sutures of linen after the plan of Witzel. The intestine is anchored to the peritoneum by two or three linen sutures in the lower angle of the incision which is closed down to the tube in the usual manner (Fig. 3) or the end of the catheter can be brought out of a small stab wound at one side of the incision, the intestine being fixed to the peritoneum on the inside by several linen sutures.

Liquid feeding may be commenced at any time and carried out for an indefinite period without danger of leakage and with a certainty that the nutritive material will pass into the assimilative tract. Leakage does not follow the removal of the tube and if it should slip out accidentally it must be replaced within twelve hours or the tract may become obliterated. All kinds of fluid nourishment are borne well in these cases—preparations of milk, eggs, meat ground fine and mixed with fluid, carbohydrates in fluid form, etc.

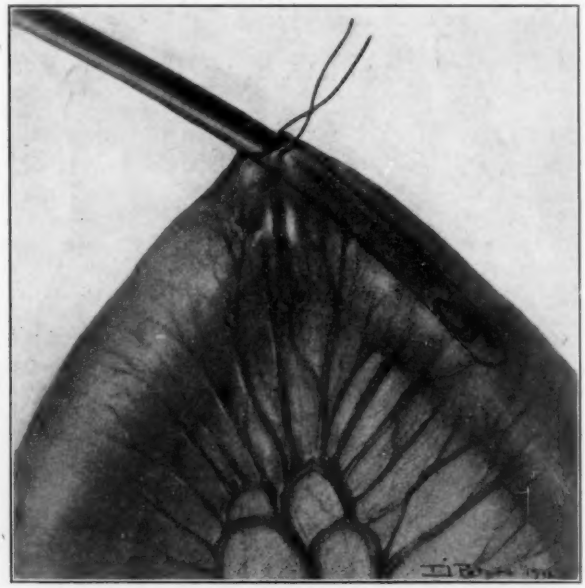

FIG. 1.—Jejunostomy: First step, showing catheter introduced into upper jejunum and fastened with a single suture.

In none of our cases have we observed subsequent ill effects from the jejunum becoming adherent to the peritoneum after the removal of the feeding tube.

In cases of esophageal and cardiac obstructions, and in diseases affecting a considerable portion of the wall of the stomach, rendering gastrostomy impossible or difficult, jejunostomy is as efficient and easier of performance than gastrostomy. It is in extensive ulceration of the stomach that jejunostomy has its greatest field of usefulness. Sometimes the stomach contains multiple ulcers, either simple or specific, in which temporary rest is indicated and gastrojejunostomy is impracticable on account of the extent

of the disease. In these cases jejunostomy affords needed rest to the stomach, a condition, it is true, which can be brought about by rectal feeding, but this procedure can in no sense be considered a building-up process; it is useful only as a temporary expedient

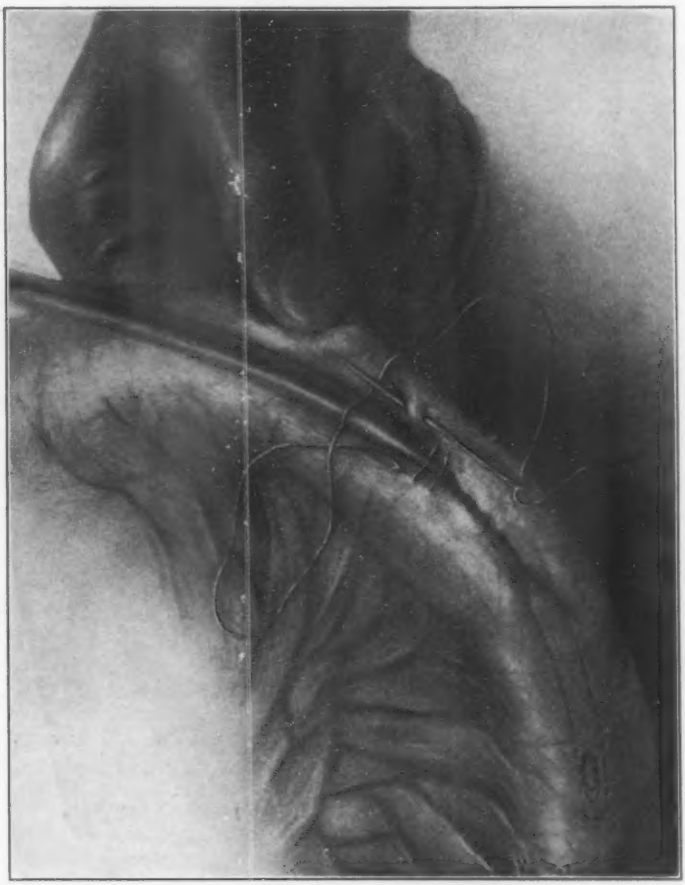

FIG. 2.—Jejunostomy: Second step, showing infolding of catheter.

to prolong life. On the other hand, jejunostomy improves the nutrition of the patient, a gain of from ten to thirty pounds can easily be made without resorting to forced feeding.

In this hypernutrition, with rest to the stomach lies the secret of the remarkable results following jejunostomy in some cases

of extensive gastric ulcers. One sometimes finds a large ulcer of the stomach so adherent and extensive and situated so high in the body as to render gastrojejunostomy not only hazardous, but open to the serious objection that it must be made distal to the ulcer. In such cases jejunostomy is a rational procedure, and even if the ulcer be malignant in character the method furnishes a useful means of palliation.

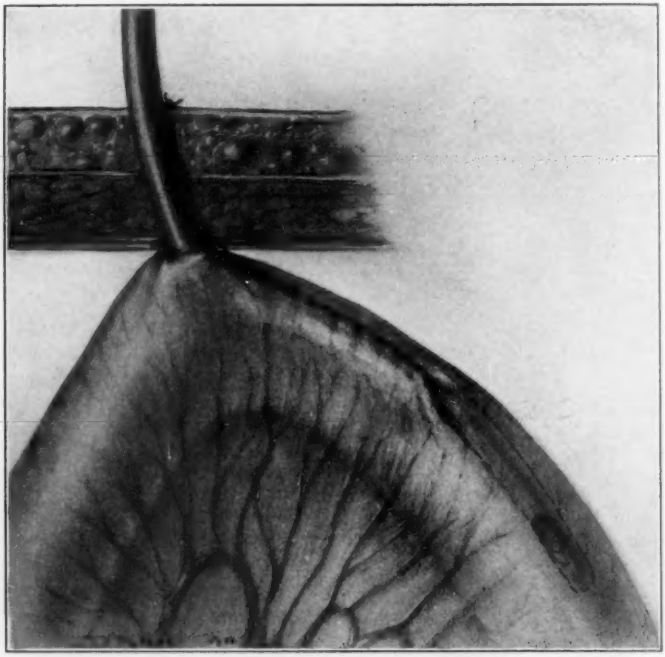

FIG. 3.—Jejunostomy: Third step, operation completed; jejunostomy fastened to abdominal wall.

Another indication for jejunostomy is in the treatment of accidental injuries to the cancerous stomach during exploration. Somewhat over a year ago while exploring a stomach which contained a large ulceration in the vicinity of the pylorus (I was trying to determine whether it was benign or malignant) I accidentally made a perforation into the foul crater of the ulcer which proved to be malignant and wholly inoperable. In attempting to repair the injury complete separation through the cancerous mass occurred. Gastrectomy was out of the question and the state of the tissue prevented the possibility of closing both ends and doing gastrojejunostomy, I therefore, reunited the cancerous tissue as best

I could with interrupted chromic catgut sutures, many of them tearing out as the tissue would not stand the strain. I then formed a sling about the line of union in the stomach with omentum, holding it in place with catgut sutures. A few rubber-tissue drains were placed about the injured stomach and jejunostomy performed. The patient was fed two months by this means. There was considerable foul discharge along the drainage tract which lasted several weeks. The catheter was removed in eight weeks, the patient gained thirty pounds in weight and lived more than a year without gastric distress and in great comfort. There was no return of obstructive symptoms in this case, although before operation the obstruction was so marked that the patient was in a chronic state of starvation and cachexia. We have observed two similar cases.

It sometimes happens, after the excision of a large ulcer of the stomach, that the organ is not in a safe condition to hold food. This is especially true of large ulcers of the posterior wall. The patients are often so debilitated that improvement in nutrition is almost a necessity in order to obtain union. Jejunostomy furnishes an easy, safe manner of giving the stomach rest during the healing process, the patient is relieved of the danger of leakage and adequate nourishment is maintained.

I am indebted to Dr. Clairmont, of Vienna, for calling my attention to the value of jejunostomy, and subsequently my interest in the operation was increased by Dr. Frank Billings, who advocated the operation on theoretic grounds, grounds which have proved correct in our clinical experience.

NOTE.—Since writing the above my attention has been called to an article in the *British Medical Journal*, January 6, 1912, by Mr. Mayo Robson in which he advocates jejunostomy in conditions of the stomach not amenable to other methods of treatment, and gives a *resume* of his cases.

PANCREATIC LYMPHANGITIS.

BY JOHN B. DEAVER, M.D.,

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INSTRUCTOR IN SURGERY, UNIVERSITY OF PENNSYLVANIA; ASSISTANT SURGEON, UNIVERSITY HOSPITAL.

"THERE is an immense amount of sepsis in medicine." These words of an eminent clinician convey an appreciation of the preponderating role played by infection in the production of path-

ology. The effects of infection are often obscure and far reaching, for they are indirect as well as direct, manifesting themselves at a distance as well as at the primary site, revealing their ravages at a time remote as well as immediately, and it is not too much to say that if we could attain the ambitious ideal of Pasteur of causing all the infectious diseases to disappear from the earth, that along with the so-called infectious diseases would go such a large proportion of diseased conditions, now but little considered in this connection, that our works on pathology could be condensed into the size of a primer and our occupation would be all but gone.

Still, it is our chief task to unravel the devious ways of infective disorders, since it is only by a thorough understanding of their etiology, clinical course and effects, that we may hope to palliate or cure and ultimately to prevent them. For this reason we offer this brief suggestion as to the origin of certain pancreatic swellings and inflammations.

That pancreatitis, both acute and chronic, is in practically every instance due to microörganismal invasion will hardly be denied, even though it be admitted that in certain instances systemic intoxications due to alcohol, syphilis, tuberculosis, or other unknown circulating toxins may be productive of degenerative lesions which may be classed as chronic pancreatitis. There is also reason to believe, on the basis of clinical and experimental evidence, that the retroinjection of bile into the pancreatic duct may set up acute pancreatitis of the most severe type even in the absence of microorganisms. Hlava's experiments show that the same result may follow the injection of gastric juice into the duct, though there is no evidence that this ever occurs clinically. Even in these cases infection has been demonstrated to follow quickly in the wake of the aseptic injury, and under conditions as they are found in the body, infection must practically always precede or go hand-in-hand with the entrance into the duct of any foreign material. Giving these considerations all due prominence, it still remains an incontrovertible fact that pancreatitis is for all practical purposes a consequence of infection, our conclusion being based not only upon analogy, but upon cultural examinations in acute inflammations, upon its frequent association with frankly infective processes in neighboring viscera, and upon the microscopic evidence of infection in the gland itself.

The chief point at issue is the method by which infection gains entrance to the gland. There are the following possible portals of entry: (1) The systemic circulation, through the arteries of supply. (2) By contiguity from adjacent viscera. (3) Ascending infection of the duct from the biliary tract or duodenum. (4) Through the lymphatics.

1. There is but little evidence in favor of infection by way of the circulating blood. The frequency of bacteremia and pyemia and the rarity of involvement of the pancreas in these conditions establishes our belief in almost complete immunity of the pancreas against serious infection through the blood stream. The clinical history of cases who are the subject of pancreatitis also furnishes no warrant for a belief in this mode of origin. When it does occur it must be classed as a rare exception.

2. Instances are not rare in which the pancreas is involved in inflammation by direct extension from a neighboring organ. The most frequent example is the chronic perforating ulcer of the stomach or duodenum which eventually makes its bed in the pancreas. In such cases the pancreas protects itself by granulation tissue and cellular infiltrate, and may suffer no general injury. In other instances it may show acute or chronic lesions more or less widespread. Bartels has reported a case of acute hemorrhagic pancreatitis associated with perforated duodenal ulcer, which is doubtless of this nature. Infection of the gland by direct contiguity is, however, relatively uncommon, the mechanism of which is simple, but fails to explain the great number of cases of pancreatitis which are now being recognized.

3. The great volume of discussion concerning the origin of pancreatitis deals with the noxious influences that may be brought to bear by way of the ducts of the gland. To stenosis or occlusion of the ducts, the injection of biliary or duodenal contents, and ascending duct infection have been assigned the chief and almost the sole part in the production of acute pancreatitis and the interlobular form of the chronic disease. Experimentally acute and chronic disease have been reproduced both by infection and by injection of the ducts. Clinically our attention has been centred upon the association of the disease with disease of the bile passages, and it has been natural to suppose that in the association of terminal facilities of the common bile duct and the pancreatic duct we have a ready means and explanation of infection of the pancreas, the affections of the latter being quite commonly accepted as being secondary to disease of the biliary tract in the majority of instances.

There are certain objections to this facile explanation. First, it is observed that the pancreas is often diseased without demonstrable disease of the biliary apparatus. In the senior author's experience about one-third (36 per cent.) of all instances of chronic pancreatitis, and 25 per cent. of acute cases were not accompanied by demonstrable disease of the biliary passages. Most investigators have laid stress upon the more frequent association of pancreatitis with gallstones, giving almost no attention to the minority of cases in which not only gallstones but even other evidences of biliary tract infection were absent. Truhart alone found that a smaller percentage of patients with acute pancreatitis had gallstones

than those who died from other causes. Desjardins supporting the belief in ascending infection, attempts to explain this by assuming that the same infection gaining entrance into the ducts of the biliary or pancreatic system may cause at one time cholangitis, or at another, inflammation of the duct of Wirsung. This is merely an assumption unsupported by clinical evidence.

A strong objection to the similarity of causes of the two conditions is their lack of parallelism according to sex. Nothing is better established than the preponderance of cholelithiasis among females, the ratio roughly being about three to two. In the series of cases of pancreatitis above mentioned the ratio was reversed, and though percentages differ, the experience of operators the world over shows that males are much more prone to this disease than are females. This fact, while not in itself conclusive indicates that probably we have laid too great stress upon the similarity of origin and intimate relations of pancreatitis and biliary disease, and directs the attention to further inquiry into the method of origin of pancreatic affections. This additional factor and one which has received altogether less attention than it deserves, we believe to be:

4. Infection by way of the lymphatics. The validity of this explanation depends upon the existence of primary infectious processes so situated with reference to lymphatic vessels that the infection may more or less readily reach the pancreas through these channels.

Of the four great sources of intra-abdominal infection, the appendix, the Fallopian tubes, the gall-bladder, and the pyloric region, the latter two are situated in close proximity to the pancreas. If it be shown that there exists an inter-communication of the lymphatics of the pancreas with these two danger points, from our knowledge of the behaviour of the lymphatic channels as avenues of infection it would seem not unlikely that the pancreas would suffer secondary involvement. Not only the grosser lesions of the gall-bladder and juxtapyloric region, such as cholecystitis and gastric or duodenal ulcer should be considered in this connection, but also those more frequent minor infectious processes which pathologically are termed catarrhal and clinically usually fail of recognition, except under the name of indigestion or dyspepsia. In the gall-bladder we are accustomed now to recognize extremely mild grades of inflammation of the organ by the presence of ever so slight thickening, diminution in lustre, increased opacity, or inspissation and tarry character of its contents. The same scrutiny should be directed toward the stomach and duodenum where slight thickening, local puckering, or distortion and increased opacity may reveal the present or past existence of inflammation of the wall. As evidence of previous inflammation we have observed dimpling of the wall at times suggesting the starting point of an intussusception. Again, there have been delicate adhesions easily to be over-

looked, and even less prominent is a peculiar streaked, opaque appearance affecting a very limited portion of the wall and often running off toward adjacent peritoneal attachments. Of course, the grosser cicatrices of healed ulcers are readily apparent, but these less conspicuous appearances should be noted as indicating, in many instances at least, the previous existence of interstitial inflammatory processes. Catarrhal inflammation, fissures, and even early ulcers may exist without external evidence of their presence, and yet as a portal of bacterial entry their importance cannot be denied as is shown by the enlargement of the regional lymphatic glands. To those abreast of the revelations of surgery in the upper abdomen no argument is needed to convince of the great prevalence of infectious processes, the potential gates of entry of organisms into the lymphatic paths of this region. It remains to show the intimate relationship of these efferent lymphatics with those of the pancreas, and our clinical basis for believing that pancreatitis may originate in this way.

ANATOMICAL CONSIDERATIONS. The brilliant study by Bartel of the lymphatics of the pancreas must be consulted in order fully to appreciate their anatomy. This work was carried out both upon animals and human subjects, and from it we draw freely. The pancreas, unlike certain other organs, possesses no great hilus through which pass the afferent and efferent blood and lymph vessels. The lymphatics emerge at various points along its surface, and run to the regional glands, to neighboring trunks or plexuses. This arrangement should be especially noted, as it leads to a very important deduction.

It may be pointed out that by reason of its retroperitoneal situation it bears a close relation to the thoracic duct and to many trunks which empty into it from the visceral and parietal lymphatics of the abdomen. As regional glands of the pancreas, demonstrated by the Gerota method of injection, Bartel describes: The pancreaticosplenic and superior pancreatic, the superior gastric, hepatic, pancreaticoduodenal (anterior and posterior), mesenteric, mesocolic, inferior pancreatic, and peri-aortic (Figs. 1 and 2). Once he demonstrated a direct communication between a pancreatic lymphatic and the lumbar trunk, but regards this as a rarity. He never succeeded in showing a direct communication with the thoracic duct, though this had been previously described by Hoggan. To all the above groups of glands the pancreas sends lymphatic branches which may anastomose in the cellular retroperitoneal tissues with the lymphatics from the stomach, duodenum, spleen, liver, gall-bladder and bile ducts, colon, and even the left suprarenal. Probably still other intercommunications exist which were not demonstrated. Lymphatic borne infection from these areas, in order to reach the pancreas, must in most cases stem the efferent current from the pancreas, and force the valves. Here, as

elsewhere in the body, the lymphatics are wonderfully efficient in preventing this outcome. Were it not true, our chief organ of digestion could not escape grave damage in a large percentage of cases, and pancreatitis would become one of the "Captains of Death."

Only when the inter-communications of the pancreatic lymphatics with those of adjacent organs are most intimate, short in their course, and unprotected by intervening lymphatic nodes does peril commonly arise. This most intimate relationship Bartel has shown to exist with the lymphatics of the adjacent duodenum, and more recently Franke has demonstrated that the same is true of the lymphatics coming from the gall-bladder

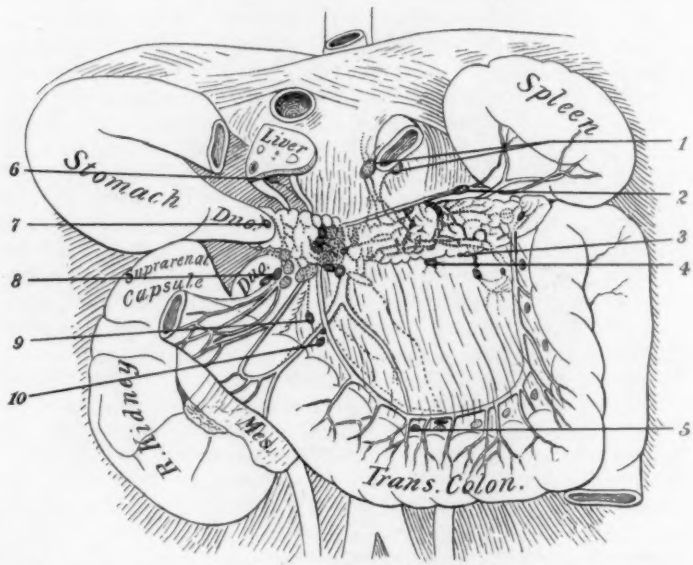

FIG. 1.—Prematurely born male child. 1, superior gastric lymph nodes; 2, superior pancreatic lymph nodes; 3 and 4, inferior pancreatic lymph nodes; 5, mesocolic lymph nodes; 6, hepatic lymph nodes; 7 and 8, pancreaticoduodenal lymph nodes; 9, mesenteric lymph nodes; 10, mesocolic lymph nodes. (After Bartel.)

How closely the finer lymphatics of the adjacent duodenum are applied to the pancreas can be appreciated by the accompanying sketch of an actual preparation (Fig. 3). The lymphatics seen upon the surface inosculate with others coming from the pancreas. In lymphatic vessels there may be a reversal of the lymph current in the effort to establish collateral circulation about points of thrombolymphangitis, and thus infection may be carried into the pancreas. An infection which is sufficiently severe would not even wait for a

reversed lymphatic current, but could ascend rapidly, involving not only lymphatic structures, but the surrounding tissues as well. Such a severe infection might well cause acute hemorrhagic pancreatitis by causing necrosis of the pancreatic tissue, liberating the active ferments, and eroding the bloodvessels. Bartel was acute enough to suggest on the basis of these observations, that pancreatitis might originate in this manner, but had no clinical material to support his inference, which received scanty consideration. Arnsperger has more recently, on clinical grounds, offered a similar

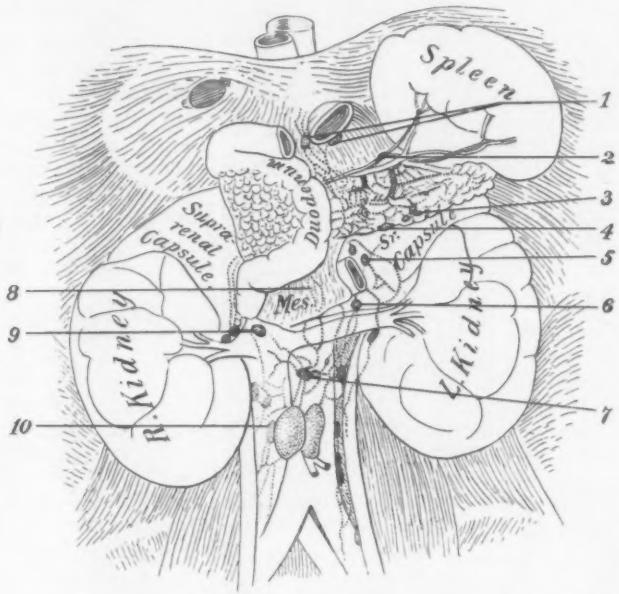

FIG. 2.—1, Superior gastric lymph nodes; 2, superior pancreatic lymph nodes; 3 and 4, inferior pancreatic lymph nodes; 5 and 6, left paraaortic lymph nodes; 7, preaortic lymph nodes; 8, from the mesenteric lymph nodes; 9 and 10, right para-aortic lymph nodes. (After Bartel.)

suggestion with reference to the association of pancreatitis and gall-bladder disease, and this view receives support from the work of Franke, who showed, also by Gerota injections, that the lymphatic vessels of the gall-bladder run to glands which lie to the left of the head of the pancreas near the choledochus, and on the right near the hepatic artery. On the way the greater part of both paths pass a gland at the neck of the gall-bladder. This gland is not, however, constant in occurrence. By injection from the gall-bladder he has filled a plexus of lymph vessels situated upon the posterior surface of the head of the pancreas. The intrinsic lymph

vessels of the pancreas run in the interlobular septa, and it is here that the effects of this type of inflammation should be most mani-

FIG. 3.—Duodenum and pancreas of newly-born dog, injected to show lymphatics. In many places an intimate connection exists between the pancreatic and duodenal lymph syatems. (After Bartel.)

fest. This corresponds with the fact that the form of pancreatitis usually associated with inflammatory lesions of neighboring viscera

is the interlobular type, as contradistinguished by Opie from the interacinar sclerosis which appears to bear no such relation to local inflammation. Of itself, however, this observation does not speak for a lymphatic origin of pancreatitis since the pancreatic blood-vessels and ducts also run in the interlobular tissue, and if inflamed would produce interlobular sclerosis. A most suggestive point is the following: It is observed by all surgeons that the earlier forms of pancreatitis found at operation in connection with the gall-bladder or duct disease involve only the head or perhaps only a portion of the head. In other words, *the distribution of inflammation corresponds to the lymphatic distribution*, which, as mentioned above, is irregularly segmental. It does not correspond to the duct distribution, which ramifies by dichotomous division from the main accessory ducts. It is most difficult to understand on the theory of duct-borne infection why the gland should not more often be symmetrically involved, while if the lymphatic method of transmission be granted this phenomenon is readily explainable. Again, the swellings of the head of the pancreas which are so frequently encountered by the abdominal surgeon must be different from the varieties of chronic pancreatitis described by the pathologist. Kehr, on clinical grounds, has surmised that such a distinction exists. Chronic pancreatitis, which is characterized by interlobular or interacinar deposits of fibrous tissue, is no more curable than is chronic nephritis, or cirrhosis of the liver. On the other hand, it is characteristic of the pancreatic swellings associated with biliary disease to subside with the subsidence of the biliary infection. This swelling must, therefore, be due to edema, congestion, and absorbable infiltrates. Its subsidence on cure of the primary infection would be analogous to that which occurs in the treatment of primary foci of lymphatic infection elsewhere in the body. It may be urged by those who advocate ascending duct infection that the disappearance of pancreatic involvement occurs as the result of the cessation of reinoculation with infected bile, and it is true that of itself this observation does not speak for either mode of infection. In many of these cases, however, Nature attempts by her own method of injections to point the course of events. In this form of pancreatitis operated upon at the fortunate time it is possible to demonstrate the chain of infection; infected gall-bladder, enlargement of the cystic gland, enlargement of the glands at the head of the pancreas, and swelling of the regional lymphatic distribution of the pancreatic head. Since we have been looking for these glands it is remarkable how constantly they have been found in this condition. One which appears to be especially constant in position and enlargement is situated just to the right of the choledochus, where it passes beneath the duodenum.

We have spoken of this condition chiefly in connection with the gall-bladder because it is here most easily capable of recognition,

but it is our belief that lymphatic infection from the duodenum may play a considerable role in chronic pancreatitis. Not a few instances of the association of duodenal ulcer and pancreatitis are on record, while catarrhal duodenitis may also contribute a share. The greater frequency of duodenal disease in males would correspond with the sex incidence of pancreatitis, which is not true of biliary disease as previously mentioned.

It is possible that still other organs may at times furnish infection to the pancreas, but the biliary tract and the duodenum seem to be the most probable primary sources on anatomical, clinical, and pathological grounds.

To this early and remediable stage of pancreatic inflammation, in order to distinguish it from the advanced types commonly designated as chronic pancreatitis, it would be well to apply the term pancreatic lymphangitis. The reasons why pathologists have failed to recognize this condition are probably to be found in the fact that the postmortem lesions are slight and can easily be confused with that autodigestion of the organ which ensues at once after death, and often renders examination of its tissues unsatisfactory, and also because the clinician has not previously called attention to the necessity for search for the minor pathological changes. Moreover, the material has been small since it is essentially a process of living pathology. It is not of itself an immediately fatal disease. It may subside with the removal of the primary source of irritation as above pointed out, and when of long standing or frequent recurrence it leads to the deposit of interlobular fibrous tissue, in other words to chronic interlobular pancreatitis.

At the present time the diagnosis can be made only by the surgeon who can inspect and palpate the gland. The treatment is that of the primary focus of infection whether cholecystitis, cholelithiasis, ulcer, or duodenitis. In cholecystitis cholecystostomy is the operation of choice. A gall-bladder damaged to the extent that recovery and permanent cure are doubtful, should be removed. When the pancreas presents marked alterations cholecystoduodenostomy is superior in its results, though the mortality of operation should decide in favor of a simpler procedure unless there are clear indications for its performance.

For the evidence of this condition it is necessary to go to the operating table. We have not yet been fortunate enough to encounter a case complete in ante- and postmortem detail. The following case is cited to show the extent to which the disease may progress. The pathology is too far advanced and the etiology too obscure to prove its lymphatic origin in this instance. The glandular involvement, however, is similar in many respects to that observed by us in a considerable number of earlier cases where the considerations just mentioned lead to the belief in a lymphatic origin.

D. G., aged thirty-seven years. Admitted to the German Hospital, January 6, 1912. Died January 19, 1912.

Chief complaint, cramps in upper abdomen, radiating into flanks and to right side of back.

Family History. Mother died of tuberculosis, otherwise negative.

Personal History. Tea, coffee, and tobacco in excess; alcohol in moderation; denies venereal diseases. In March, 1910, he was admitted to the Pennsylvania Hospital suffering with pain beneath right costal margin radiating to the back. The pain was worse two or three hours after eating. Constipation was a marked symptom, and he had lost considerable weight. At operation cholecystitis was found and cholecystotomy performed. In July of the same year he was again operated upon for recurrence of symptoms and cholecystoduodenostomy performed. In June, 1910, he was again in the hospital with the same symptoms plus jaundice, chills, and fever. Cholelithiasis was diagnosticated, but he was not operated upon. In November, 1910, after intermittent attacks resembling those of stone in the common duct he came to the German Hospital and was again operated upon. Chronic pancreatitis, chronic cholecystitis, and a stone in the common duct were found. The stone was forced into the duodenum. The cholecystoduodenostomy opening had become obliterated; cholecystoduodenostomy performed. Recovery was uneventful, and he was improved for a few weeks, when his trouble recurred. More recently he has been having chills, fever, and jaundice.

Physical Examination. A poorly nourished man of nervous appearance. Skin moderately jaundiced. Head and chest negative.

Abdomen: Scar of old incision through upper right rectus. In this region and in the epigastrium there is marked tenderness on pressure, but no rigidity or mass.

Urine shows a very faint trace of albumin, and a few hyaline casts, otherwise negative.

Blood: Hemoglobin, 73 per cent.; erythrocytes, 4,050,000; leukocytes, 7300; polymorphonuclear neutrophiles, 73.5 per cent.; lymphocytes, 26 per cent.; eosinophiles, 0.5 per cent.; coagulation time, 8 minutes.

Stool: Dark green and practically non-odorous; fluid; neutral reaction; a trace of bile, occult blood positive to benzidine and guaiac tests. Azotorrhea and steatorrhea marked.

Cammidge reaction positive.

Operation, January 13, 1912. Dr. Deaver. Ether anesthesia. Excision of old scar in upper right rectus region; adhesions of stomach and right lobe of liver to incision; hepatic flexure of colon adherent to right lobe of liver; great omentum adherent to lesser omentum; small whitish tubercles found on small intestines; some adhesions between ascending colon and parietal peritoneum; liver covered with a few whitish nodes which are subserous;

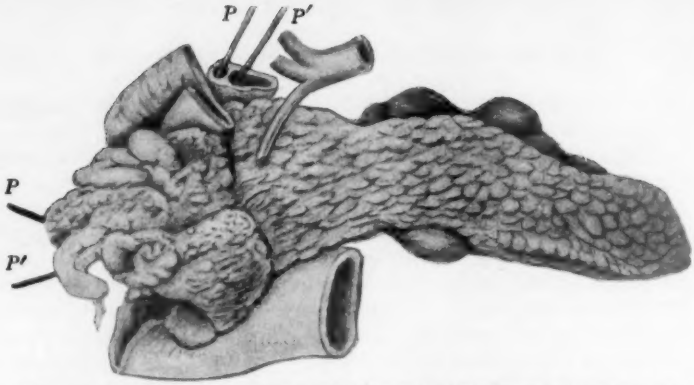

FIG. 4.—Anterior view of pancreas: P and P', probes in choledochus and fundus of gall-bladder near gastro-enterostomy opening. Celiac axis adherent to upper margin but not in true relation. The nodules adherent to the head and upper and lower margins of the pancreas are hyperplastic lymph nodes.

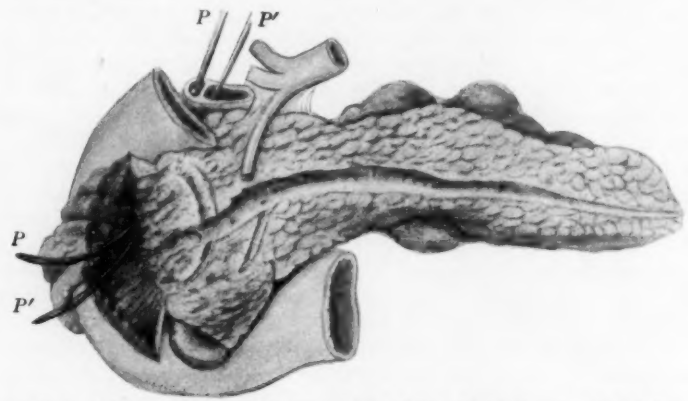

FIG. 5.—Anterior view with duct of Wirsung opened throughout its length and a window in duodenum.

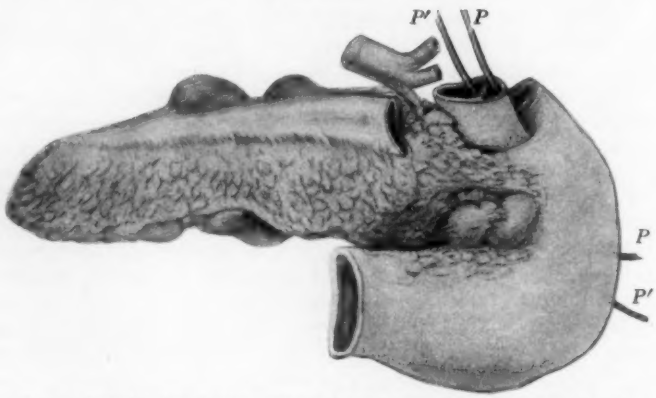

FIG. 6.—Posterior view showing enlarged posterior pancreaticoduodenal lymph nodes.

old cholecystoduodenostomy still patulous; head of pancreas enlarged and hard; felt semicystic; foramen of Winslow, though occluded, was forced open; small glandular enlargement at junction of the supra- and retroduodenal portions of the common duct; head of pancreas opened by inserting scissors and withdrawing them opened (Hilton's method). Much hemorrhage followed this; was controlled by a piece of selvage gauze and one suture of iodine gut; duodenum incised and old cholecystoduodenostomy proved patent; much bile-stained material escaped from opening; head of pancreas seemed to be obstructing the duodenum posteriorly from pressure; duodenum closed with catgut baseball suture and a Lembert of linen thread; pancreas at site of incision sutured to parietal peritoneum; one cigarette drain anterior to gastrohepatic omentum; one rubber tube at foramen of Winslow; another rubber tube anterior to this and wound closed in layers.

Following the operation there was profuse brownish drainage from the wound. The jaundice disappeared. He became more emaciated and gradually weaker and died January 19, 1912.

Autopsy through the incision permitted removal of the pancreas and adjacent tissues. The head of the organ was somewhat smaller than normal, and appeared necrotic. The selvage gauze in the pancreatic wound had ulcerated into the duodenum at the site of the ampulla of Vater. A fistula existed at the site of the exploratory incision into the duodenum. A striking feature was the occurrence of many enlarged lymph nodes along the upper margin of the pancreas. A few were present along the lower border, and in relation to the head both anteriorly and posteriorly where embraced by the duodenum.

Microscopically the tail of the pancreas was the seat of a moderate grade of interlobular sclerosis. The parenchyma was mostly degenerated, probably, chiefly due to autodigestion. The islands of Langerhans were intact. The head of the organ showed marked interlobular pancreatitis and an acute suppurative process superimposed. The parenchyma was necrotic. The islands of Langerhans persisted but were involved in the acute necrotic process. A section made through a small villous patch at the junction of the gall-bladder and duodenum (at the cholecystoduodenostomy opening) showed a marked glandular proliferation which had begun to invade the deeper tissues. The process had not extended more than 0.5 cm. from its starting point, and no metastasis could be demonstrated. The lymph nodes showed chronic and acute hyperplastic lymphadenitis.

We append this history in outline with illustrations because it shows the situation and appearance of the lymph nodes in many cases of pancreatic lymphangitis. It may well be stated in connection with this case that the lymphatic involvement was secondary to the disease of the pancreas, and indeed this is true in part at least.

We have, however, observed not a few cases in which regiona. enlargement of the lymph nodes was present without involvement of the pancreas so far as could be demonstrated. Again, they have been seen in conjunction with gall bladder and duodenal disease, the pancreas still being unaffected. Finally, in still other cases, this appearance has been associated with sclerosis and nodular thickening of the pancreas which we have come to believe represent the communication of infection to the pancreas by way of the related retroperitoneal lymphatics.

CHRONIC CHOLECYSTITIS.[1]

By J. Russell Verbrycke, Jr., M.D.,
WASHINGTON, D. C.

MacCarty,[2] from the study of 365 cases of cholecystitis in which cholecystectomy was performed at St. Mary's Hospital, concludes that the different lesions of the gall-bladder are not definite entities, but are different degrees of the same process of reaction to irritation. He divides the cases into 8 groups: (1) Acute catarrhal; (2) chronic catarrhal; (3) papillomatous catarrhal; (4) malignant papillomatous; (5) carcinomatous catarrhal; (6) chronic cholecystitis; (7) chronic cystic; (8) purulent, necrotic. Two hundred and fifty-two, or 67 per cent., of the 365 cases were accompanied by gallstones. The chronic cases showed a large proportion of stones, while the early catarrhal conditions did not necessarily occur associated with stones.

It is to the large class of mild catarrhal cholecystitis, with or without stones, that I wish largely to confine this paper. How many people are thus affected is becoming more and more appreciated, due largely to the work of such men as Moynihan, the Mayos, Deaver, and a number of German surgeons and pathologists. By some the number of individuals having gallstones is overrated, but the great mass of physicians have been slow to grasp the real frequency of cholecystitis with or without cholelithiasis.

In Germany, as a result of the study of hospital autopsies, it has been estimated that of those dying in hospitals from all causes, 10 per cent. have gallstone and 13 per cent. cholecystitis. In England and this country it has been claimed by some that 6 per cent. have gallstone disease. Wm. J. Mayo[3] is more conservative and his conclusions are more reasonable. He thinks that 3 per

[1] Read before the Georgetown Clinical Society, November 6, 1911.
[2] Jour. Amer. Med. Assoc., liv, 2004.
[3] Ibid., lvi, 1021.

cent. is probably in excess of the truth and that one-half of 1 per cent. is a fairer estimate of the frequency of gall-bladder disease at all ages, though he says that 5 to 8 per cent. of women and 2 to 4 per cent. of men have gallstones after the age of fifty. Of 1224 women operated upon for uterine fibroids at St. Mary's from 1901 to 1910, 7½ per cent. had gallstones.

Waterhouse[4] believes that the majority of the fat, flatulent women seen at the clinics so constantly, without obtaining much relief, are really gallstone sufferers.

There is little doubt of the correctness of the theory that gallstones are produced through the action of attenuated bacteria which produce a desquamation of the epithelium of the gall-bladder, this serving as a nucleus upon which the cholesterin and other salts are deposited. Bacmeister[5] holds that simple stagnation of the bile, *per se*, may cause cholesterin to crystallize out and build up one or more stones by radial formation without previous inflammatory processes being present. But, he says that after the stone is formed it may produce irritative inflammation.

Moynihan[6] has described a condition in which fine grains of calculous material are embedded in the mucosa. The wall of the gall-bladder may appear normal, being thin and pliable. Particles of the grit are also found in the bile, but no stones.

Why cholecystitis should develop in some individuals and not in others cannot be fully explained at present. Bacteria in health can be, and are, excreted through the gall-bladder. Some conditions must be present favorable to their development in the gall-bladder. How does excessive intestinal putrefaction influence the production of cholecystitis? Is it because of an increased number of bacteria carried by the portal vein to the liver in this condition? Why do gallstones and obesity so often come together? How is it that cholecystitis occurs so frequently in pregnancy and that a preëxisting stone often ceases to be quiescent at that time? These and other questions can only be answered tentatively. The influence of sedentary life and overeating is somewhat easier to explain.

A cholecystitis is probably nearly always present before stone formation, but after the appearance of the stone each condition is made worse by the other. The early symptoms are due to the inflammation whether a stone is present or not.

Gall-bladder disease is a hard malady to diagnosticate, hardest in the first stages before stones have formed and in the cases where a mild catarrhal process has continued for years without producing colic or jaundice. Such a condition may persist for two to six years or even longer. Stones tend to stay in the bladder. After colic has occurred or jaundice is present the diagnosis is easy,

[4] Lancet, May 8, 1909.
[5] Münch. med. Woch., May 11, 1909.
[6] Annals of Surgery, December, 1909.

but much valuable time has been lost and cases should not be allowed to progress thus far. Later the diagnosis again often becomes difficult if frank colics have ceased and the symptoms are due to the results of a previous cholecystitis and stone, adhesions, etc., as expressed by Morris—"cobwebs in the attic of the abdomen."

Until a few years ago cholecystitis or gallstones were not diagnosticated until the occurrence of colic or jaundice, symptoms of complications which render all treatment more difficult. Now, early cases are recognized, most often by the specialists and the surgeons to whom, principally, is due the credit for our understanding of the symptoms during the first stages, previously "innocent gallstones."

As before mentioned, the writer is not in accord with the expressions of a number of those who claim that the early diagnosis of cholecystitis is easy. It is difficult. Mayo states that questioning those in whom gallstones were found at operation for other troubles practically always resulted in obtaining a history of the earlier symptoms of gallstone dyspepsia. However, it is decidedly easier after we know a condition is present to obtain a satisfactory corroborative history than it is to make such a diagnosis before. Another point, in the average patient it is next to impossible to obtain an accurate history of the beginning of a mild chronic trouble of slow progress. Patients have either paid little attention to the mild dyspeptic symptoms or have forgotten what impressed them at the time.

We are told that the gall-bladder has its own type of digestive disturbance (Coleman). To this I can hardly subscribe. The dyspepsia of cholecystitis more often than otherwise is not characteristic. The most that we can say is that some of the symptoms are decidedly suggestive, and, if we can rule out disease of the stomach and appendix, or, if we can find tenderness over the gall-bladder, then we can be more certain. Some writers differ entirely in describing certain symptoms, as for instance the time of occurrence of pain and the effect of food.

I shall mention some of the symptoms of mild cholecystitis or gall-bladder dyspepsia as advanced by different observers. Moynihan's "inaugural symptoms" are slight pain, not severe, but a discomfort, most often in the epigastrium, but not well localized; uneasiness; flatulence; ill-defined sensation of tightness; weight and oppression generally referred to the stomach after meals, which is usually relieved by belching; more severe pain which may radiate to the right shoulder. He describes a feeling of coldness and a slight shivering, and again a sudden short catch in the breath. Still other symptoms are drowsiness, inability to concentrate the mind and a sense of weight and fulness in the head. His confirmatory signs are tenderness on deep inspiration to thumb pressure

under the edge of the ribs, or dorsal tenderness and skin hyperesthesia over the area corresponding to the gall-bladder zone, the eleventh dorsal vertebra to the first lumbar and from one inch to the right of the spine to the posterior axillary line.

The above symptoms may come on at night together with palpitation of the heart. Here it may be noted that Babcock[7] has reported 13 cases in which myocarditis was so severe that the gall-bladder trouble was entirely overshadowed by the heart symptoms. Others also have noted the marked heart symptoms.

Graham, quoted by Coleman,[8] distinguishes four stages in the development and symptomatology of gall-bladder disease. In the first stage there are symptoms of mild disease of the stomach, irregular attacks of indigestion accompanied by gas formation and a feeling of epigastric constriction or tightness. There is discomfort, sudden in onset and frequently accompanied by chilliness, belching, regurgitation or vomiting. The chief characteristics of these attacks are their suddenness, irregularity and mildness. In the second stage pain occurs which may be dull or more severe and in located under the right costal arch. It may be aggravated by food, sudden exertion or deep inspiration. The attacks are evanescent in type and on their disappearance the patient enters upon a period of good health. The third stage is that of typical gallstone colic. Here the diagnosis is easy. The surgeon sees the majority of patients first in this stage. The fourth stage represents chronic gall-bladder disease with complications of adhesions, obstructions of the common duct, pancreatitis, cancer, etc. In this connection I may remark that pancreatitis sometimes appears before the fourth stage is reached. I have had one patient exhibit symptoms of beginning pancreatitis before she had experienced a single attack of real colic and another has had but one attack which was unaccompanied by obstruction or jaundice.

Sherren,[9] basing his report on 43 patients with gallstones, considers the special points in the dyspepsia due to biliary trouble to be the associated flatulent distention, the frequency with which the patient is awakened at night, the non-relief of pain by taking food, and the relief by vomiting if this should occur, though it is unusual. He says that abdominal examination may settle the matter, as there may be a distended gall-bladder which may be felt or a slight tenderness under the right lower ribs may be present.

Wm. J. Mayo, in speaking of cholecystitis without stones, says that there is usually an habitual tenderness in the region of the gall-bladder. He says that as a rule colic is not a prominent symptom and, if present, is not as severe in character as that accompanying stones. He speaks of the presence of mild constitutional symptoms, lassitude, and other signs of mild toxemia, there being some-

[7] Jour. Amer. Med. Assoc., lvi, 887.
[8] Ibid., lii, 1904.
[9] Lancet, April 1, 1911.

times for months a slight evening temperature without loss of weight. He describes the two most frequent forms of non-calculous cholecystitis as (1) a distended thin-walled gall-bladder containing several ounces of colon-infected bile, often of foul odor; (2) a gall-bladder with walls much thickened and white, often containing a thick, tarry material.

Whenever I see a stout woman, around or past middle age, with dyspeptic symptoms I think of gall-bladder disease and do not dismiss it from my mind until I am certain that her symptoms are due to other causes. A previous history of typhoid is significant. The points upon which I would lay particular stress are the following: Mild dyspeptic symptoms which are neither constant nor periodic, but appear suddenly at irregular intervals and disappear in like manner; the absence of a gross dietary indiscretion or if an attack has followed eating of some indigestible substance, as is sometimes the case, the absence of attempts on the part of the stomach and intestines to rid themselves of the offending substance by vomiting or purging; the presence of considerable flatulence; discomfort or mild pain not strictly localized, but confined to the upper part of the abdomen; the frequent appearance of this pain at night; and often a subicteric hue to the skin. These are the most characteristic symptoms of gall-bladder dyspepsia, but I do not feel warranted in making a diagnosis until I can obtain some tenderness in the gall-bladder region or perhaps only a fleeting rigidity of the right rectus, and until I have thoroughly examined the urine, stomach contents and feces to rule out other conditions. Some reflex disturbance will actually be found in the stomach of a number of patients. Lichty after gastric analyses in 156 gallstone patients deduced the fact that 75 per cent. of all gallstone cases have secretory disturbance and two-thirds of these have hypersecretion. Disturbances of motility exist in nearly the same proportion.

I have thought that an aid to the diagnosis of pathological changes in the bile might be found in the use of the Einhorn duodenal pump and the aspiration from the duodenum of either of the two sorts of bile described by Mayo as occurring in this condition, but as yet I have not had occasion to try this.

I have purposely spoken of the diagnosis of cholecystitis and not of cholelithiasis since the latter diagnosis cannot be made in the absence of colic. It should be stated that inflammation of the gall-bladder is present and probably one or more stones. Kehr, after performing 1668 gallstone operations says that only in exceptional instances is it possible to state positively the presence of gallstones.

The conditions described by various observers, which must be differentiated from cholecystitis, may be briefly mentioned and comprise, gastric and duodenal ulcer, cancer, intestinal colic, gastric neuroses, reflex disturbances from chronic appendicitis,

crises of locomotor ataxia, etc. A rare but extremely hard condition to differentiate is chronic gastromesenteric ileus. I saw such a case with Drs. Morgan and Clark. The patient's trouble had been diagnosticated by others and by us as either ulcer of the stomach or gall-bladder disease. Operation disclosed nothing but a dilated stomach and first part of the duodenum due to gastromesenteric ileus. A gastro-enterostomy has completely relieved her.

In discussing the treatment of gall-bladder dyspepsia or cholecystitis I shall confine myself to the medical side. Some there are who believe that the only medical cases are those in whom an operation is not feasible. This is the view held most frequently by American and English surgeons. They believe that patients should be operated upon as soon as the diagnosis can be made, before complications occur to render operation more difficult. Deaver, however, says that it is impossible to differentiate a stone-forming biliary catarrh from any other biliary catarrh, so it is necessary to wait until stones are actually formed before we have a basis for operation. German surgeons also are slower to operate. Kehr[10] claims that operative treatment is required more for the infectious processes and to prevent their spread to other organs than to remove the stones themselves.

There can be little doubt that cholecystostomy, or even cholecystectomy, is indicated in a number of cases, particularly long-standing cases and those of considerable severity, often accompanied by complications. Also there can be little doubt but that drainage will help or cure all cases including the earliest, whether stones have already formed or not. However, no one is anxious for unnecessary operations, and if it can be shown that in certain patients the removal of the infectious processes can be secured by other means operation would not be favored in these instances. This I will endeavor to do.

Kolisch,[11] after years of experience, is convinced that dietetic measures are probably the most important of all in the treatment of gall-bladder disease. His aim is not to attack the stone, if one be present, but to combat the inflammation in the gall-bladder. Treatment should aim to avert anything liable to further injure the already weak biliary functions, and diet should be arranged to spare the liver as much as possible and keep the intestines in good order and free from catarrhal processes. He ascribes the good effect of Carlsbad water to its effect on the intestines. The diet should contain no chemical, thermal, or mechanical irritant. There should be no overeating. Particularly to be avoided are condiments, spices, acids, raw vegetables, and fruits, fats which do not melt readily, fat and salted meats, foods prepared with yeast or fermentation, cold drinks, much albumin with extractives and

[10] Münch. med. Woch., March 21, 1911.

[11] Medizinische Klinik, April, 1910.

purins. He advocates in conjunction with the diet abstinence from rough sports for at least a year and claims as a reward permanent latency of the gall-bladder disease.

My own treatment for all early cases and mild cases, which I have used with good results even in some cases that had had attacks of real colic is something like that of Kolisch, but with added features which I think make it somewhat more reliable.

The patient is told to take, on awakening, one to two glasses of warm Carlsbad water, after which to turn on the right side for twenty minutes. On arising, if conditions do not contraindicate at the time, a few selected mild abdominal exercises are to be performed followed by a cold towel bath while standing in warm water. The brisk rubbing is followed by a glowing cutaneous circulatory reaction.

The diet is regulated so that there shall be no overeating and no irritation. It should be a well-balanced diet with, however, a preponderance of cereals and vegetables. The same things are forbidden as by Kolisch and, in addition, a still further reduction of fats. Even the more easily digested fats, as butter, olive oil, and cream, are minimized. In order that constipation may not result or be aggravated by the small amount of fat I order at bedtime 2 or 3 tablespoonfuls of albolene. This acts as a lubricant, is not digested or absorbed and is excreted from the body in the same form in which it is ingested. Albumins are considerably reduced in order to minimize intestinal putrefaction which is nearly always manifest. If a somewhat low caloric value is attained in the diet at first I consider it an advantage, rather than otherwise, if the patient is stout.

Medicinal agents calculated to reduce the inflammation in the gall-bladder, antisepticize the bile and render it more fluid, are hexamethylenamin administered several times a day up to a total of 30 grains in twenty-four hours, and bile salts 5 grains three times a day. Later both the remedies may be cut down to a smaller amount and continued for a considerable length of time. If signs of a relapse appear the doses may be temporarily increased. At the beginning of treatment if the inflammation is subacute hot poultices and complete rest in bed for several days are indicated.

I cannot speak authoratively as yet as to the permanency of the results of this treatment, but I have so far no reason to regard it with anything but confidence and, at least, worthy of a trial before operative measures are undertaken. I know that the inflammation rapidly subsides and the patient feels much better almost from the start. Again, I feel that if operation is performed this treatment will render convalescence shorter and cure more certain. Recurrence after operation is not so infrequent but that it is well to take precautions.

Several cases will illustrate the symptomatology of gall-bladder dyspepsia and the rapid subsidence of symptoms under treatment.

Mrs. M. A. T., white, aged sixty-nine years, married, seven children. Past history negative. Present illness dated back for thirty years, coming in attacks, perhaps once a month. For the last ten years she had been free from them. There was a return of the trouble the month before she consulted me. During that month she had three attacks. Her attacks consisted of pain in the pit of the stomach and at times over the gall-bladder. When severe they radiated to the back and right shoulder. She was always puffed out with gas at the time and just before the attack would belch considerably. There was considerable nausea but rarely vomiting. Her bowels were generally constipated.

Physical examination disclosed everything normal except the following: Belly large and pendulous, panniculus abundant, and recti somewhat relaxed. Moderate tenderness under right costal border but gall-bladder not palpable. Slight tenderness across the mid-epigastrium becoming less on the left side. This corresponded with the position of the pancreas. Weight was 249 pounds; hemoglobin, 85 per cent.; blood pressure, 160 mm. A diagnosis was made of gall-bladder disease. She probably had a stone at first, which was passed, as it is inconceivable that it should remain latent for ten years when attacks had been so frequent before. At the time of examination she was just recovering from one of the attacks. No jaundice had been evident. The tenderness over the pancreas without doubt indicated a low grade of inflammation in that organ. After four days of treatment all gall-bladder and pancreatic tenderness had completely disappeared and she felt perfectly well. While she has had one attack, mild in nature since, it was when she lapsed in her treatment and she has had no symptoms for the last three months.

Mrs. E. G., white, married, three children, aged thirty years. Referred to me by Dr. R. R. Walker. Family and past history were negative. She began to have "stomach trouble" seven years ago and for the two years before consulting me was much worse. During this time she gained in weight. The trouble would come in attacks at irregular intervals, toward the last coming oftener and being more severe. Her symptoms were discomfort in the epigastrium, nausea, occasional vomiting, bloating, and a feeling of flatulence, a good deal of belching and heartburn between attacks. Her pain was also often in the gall-bladder region and would at times radiate to the shoulder. It was not dependent on food and would frequently come on during the night. Her bowels were irregular.

Examination showed a stout healthy looking woman, with good color in her cheecks but a subicteric hue to the skin. The scleræ were decidedly bile tinged, but she had never had real jaundice.

All organs were normal save the following: There was a spot of moderate tenderness in the mid-epigastrium, just below the xyphoid, also, in the gall-bladder region and extending across the upper abdomen to the left side, over the pancreas. The urine showed the faintest trace of albumin and a slight excess of indican. The strong benzidine reaction for occult blood in the stool when on regular diet disappeared after a meat-free diet. The thread test for ulcer was also negative, so this condition was ruled out. A diagnosis was made of gall-bladder disease and slight pancreatitis. Five days after beginning treatment she felt and looked well. The tenderness and other symptoms had completely disappeared and she has had no further trouble in the last six months except for a slight spell while away from the city and not taking care of herself.

I shall mention the case of Mrs. F. because her condition was more acute with slight fever and a leukocytosis of 15,400. Her symptoms were about the same as those of the above mentioned patients, but more pronounced. She, also, has been doing satisfactorily. In beginning treatment in her case rest and the icebag were used for several days.

In cases of acute cholecystitis of severe type operation should be performed without delay. I would not have it understood that I favor trying this medical treatment in any case where there is danger in putting off operation.

In closing I would emphasize the following conclusions:

1. There are numbers of dyspeptics whose symptoms are due to gall-bladder disease.
2. Diagnosis should be made early and treatment instituted before the occurrence of gallstones if possible.
3. Surgery is the only treatment for a certain class of cases.
4. In all early cases, exclusive of those dangerously acute, medical treatment, according to the principles outlined above, should be tried before recourse to surgery.
5. In a number an apparent cure will be effected, particularly if no stones have formed and it is usually impossible to state whether or not stones are present.
6. The medical treatment can do no harm in those patients who have not yet had symptoms of obstruction.
7. If two attacks occur after treatment has been started there is still time for surgery, and an operation should then be performed. It is not advisable to give medical treatment further trials.
8. In those cases which do finally come to operation the previous treatment will usually have put the patient in a better condition.
9. The foregoing diet and medication should also be given in the post-operative treatment of those who require surgical intervention.

THE VALUE OF FROZEN SECTIONS IN THE TEACHING OF PHYSICAL DIAGNOSIS.[1]

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The thought of employing frozen sections in the teaching of physical diagnosis had its inception some three years ago. At that time, the writers were investigating the causation of the hydrothorax resulting from chronic valvular heart disease. Some of the bodies used for this study happened to be those of individuals who had died from pulmonary tuberculosis. In studying these sections it became apparent that by their use the gap which had hitherto existed between the pathological specimens and the interpretation of the signs produced by such a lesion in the living subject could easily be bridged. The method usually followed in teaching physical diagnosis is to demonstrate on the living subject certain physical signs, the teacher then stating, or indicating by diagrams that such and such signs indicate such and such a condition. In the instances in which it is possible for the student to see the pathological specimens from a case studied during life, much of the value is lost, because of the dissociation of the organ from its anatomic surroundings, and also, in the case of the lungs, by their collapse.

Up to a comparatively recent time, the study of anatomic relationships and minutiæ was based entirely on dissections of soft bodies. Under this system of learning anatomy it was impossible to get an *x*-ray conception of any given area of, for example, the chest. One could know what lay *immediately* under certain ribs, but when a through and through conception was desired it could be only approximately accurate, this being true of both the thorax and the abdomen, but especially of the former. The introduction of freezing and sectioning of cadavers has added to the accuracy of our conceptions, but this method, while valuable for study purposes, is open to the objection that the specimens lack permanence, and are, therefore, unfit for repeated demonstrations. Now that we have

[1] Read before the Section on General Medicine of the College of Physicians of Philadelphia, November 27, 1911.

a means of preparing and preserving our material by the use of formalin and freezing, our specimens are available for repeated study and indefinite use.

FIG. 1—Sagittal section through left apex. This section shows a posterior cavity with the remainder of the apex practically normal. The physical signs in front would be obscured by the almost normal tissue.

The art of diagnosis is, after all, the art of visualization, and in order to visualize the abnormal, a thorough ability to mentally picture the normal is essential. The groundwork of accurate diagnosis is a keen knowledge of the anatomy of the parts concerned, and the diagnostic work of a clinician can never surpass in accuracy his knowledge of the normal anatomy of the parts dealt with. While

it may be true that ultra-minute correctness in diagnosticating lesions is not necessary in, for instance, tuberculosis, in order to treat a given case correctly, still the aim of the teacher should be to impress the necessity for such correctness upon the students under his tutelage.

FIG. 2.—Sagittal section through right apex. This section shows a subpleural anterior cavity with densely infiltrated tissue below and in front. The physical signs would be most marked above and below the clavicle. The distance from the supraspinous fossa to the cavity should be noted.

One trouble in teaching the diagnosis of chest conditions is that the student comes up to that point in his undergraduate career with an anatomic knowledge largely of a descriptive character. He has acquired it by lectures and in the dissecting room, has traced the pulmonary artery into the lungs, the pulmonary veins into the heart, has seen the trachea and bronchi, has learned the order of vein, artery, and bronchus from above downward, and from before backward, and has collected other semi-useless bits of

anatomic A, B, C. But what conception does he have from personal observation of the real shape of the pulmonary apices, or of that of the chambers of the heart? What does he know of the difference between the depth of the thorax, from sternum to vertebræ, and that from nipple directly backward to rib? What true

Fig. 3.—Sagittal section through right apex. This section shows ragged communicating cavities with dense infiltration of the lower lobe. The physical signs would be given both in front and behind. The distance from the supraspinous fossa to the cavity should be noted.

idea can he have of the amount of lung that lies posterior to the anterior facies of the vertebral column? How can he know of the difference in thickness of muscle and bone between the posterior aspect of the pulmonary apex and the surface, and that between the anterior aspect and the surface? How can he have any con-

ception of the mediastinal relations of the lungs, since in order to see them he has to destroy them?

The teaching of physical signs is open to the same criticism. The instruction is either abstract, dealing with variations in the percussion note and breath sounds, or if the living body is used to demonstrate these changes, the student is working with the chest wall interposed between him and the lesion being investigated. He is required to exercise his imagination without anything (except possibly diagrams) to aid him. The tremendous gap between the physical signs on the one hand and the lesion being studied on the other is entirely unbridged. As we already have pointed out, the only aids hitherto afforded have been diagrams, and the teacher's say-so.

FIG. 4.—Frontal section in the axis of the trachea. This section shows the inclination to the right of the trachea, the relation of the right and left apices to the trachea, and the distance of the cavity in the right upper lobe from the top of the shoulder.

The study of living pathology is, of course, the ideal way to acquire knowledge of gross lesions. The next best method is to have our specimens of dead pathology as near as possible in the conditions present during life. The ravages of advanced tuberculosis can be well shown a student by placing before him a lung which is badly diseased, but this is all that can be shown.

Our experience has convinced us that the use of hardened sections

has filled this hiatus most satisfactorily. It cannot be denied that it is preferable to work from the known to the unknown, rather than the reverse, as is usually done. Applying this to the question

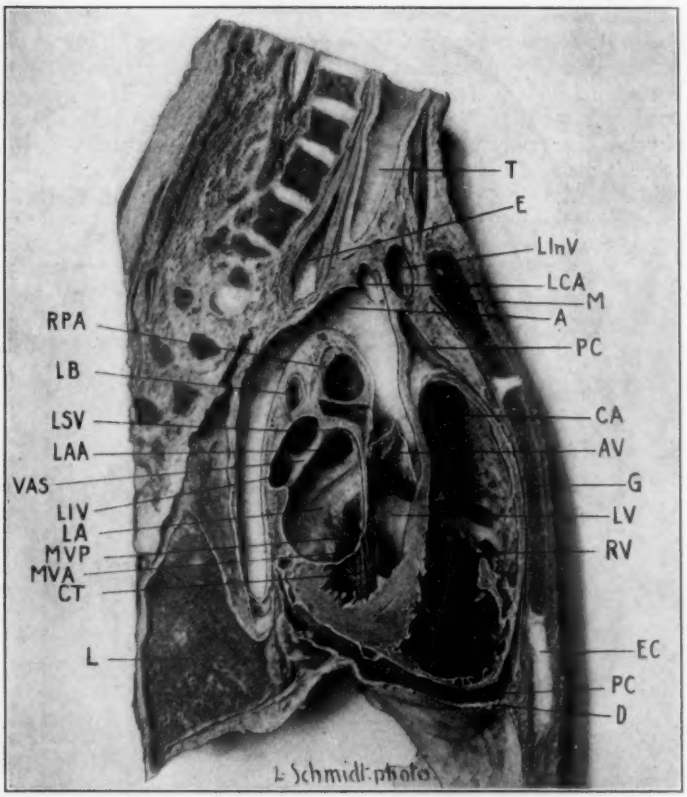

FIG. 5.—Sagittal section in the median line. This section shows the almost directly backward course of the aorta, the relation from before backward of the right ventricle, left ventricle, and left auricle, the relation of the aortic and anterior mitral leaflets, etc.: *L*, lung; *CT*, chordæ tendineæ; *MVA*, mitral valve, anterior leaflet; *MVP*, mitral valve, posterior leaflet; *LA*, left auricle; *LIV*, left inferior pulmonary vein; *VAS*, veno-appendicular septum; *LAA*, left auricular appendix; *LSV*, left superior pulmonary vein; *LB*, left bronchus; *RPA*, right pulmonary artery; *T*, trachea; *E*, esophagus; *LInV*, left innominate vein; *LCA*, left common carotid artery; *M*, manubrium; *A*, aorta; *PC*, pericardial chamber; *CA*, conus arteriosus; *AV*, aortic valve leaflets; *G*, gladiolus; *LV*, left ventricle; *RV*, right ventricle; *EC*, ensiform cartilage; *D*, diaphragm.

under discussion it would seem far more rational to show the student a section of a tuberculous lung *in situ*, and indicate what physical signs must necessarily be produced from such a lesion,

than to demonstrate a chest *in vivo*, and tell him what the signs indicate.

In teaching students the physical signs of tuberculosis at the Phipps Institute of the University of Pennsylvania, the following procedure is employed. They first examine a series of extracted

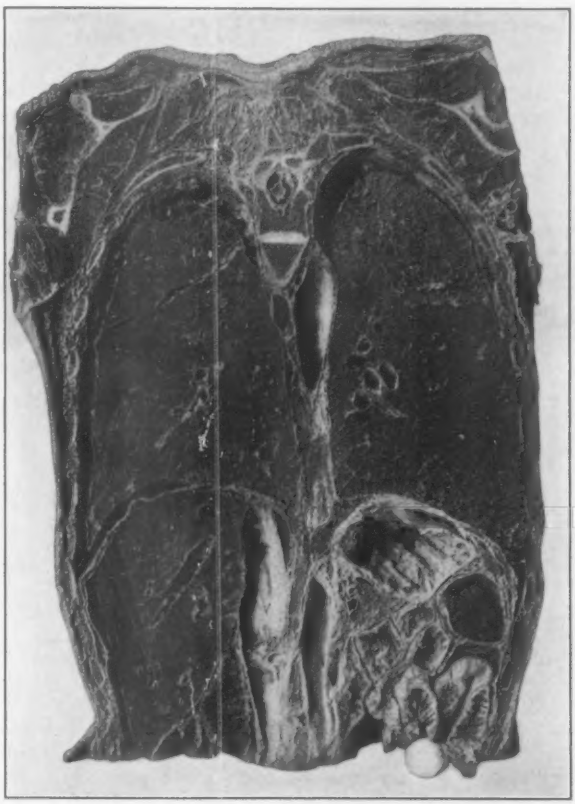

FIG. 6.—Frontal section. This section shows the distance from the supraspinous fossæ to the apices, the lower pulmonary lobes comprising the greater part of the posterior lung space, the contact of the left lung with the thoracic aorta, and the relation of the right base to the liver and the left base to the stomach.

lungs, preserved by the Keiserling method. These specimens show practically every change from that of a small cluster of tubercles, illustrating an incipient lesion, to the most destructive ravages of advanced disease. The student is then shown the hardened sections, and has an opportunity to see the cavity in relation

to the ribs, clavicle, scapula, and soft parts, and according as the cavity is small or large, anterior or posterior, and covered by a thick or thin pleura, he can be taught at once what physical signs should be produced.

The next step is to assign a case for examination, and having seen a diseased lung *in situ*, he is well equipped to form a mental picture from the physical signs. Finally, after he has charted his findings they are censored by one of the instructors, and if any point is not clear, recourse is again had to the hardened preparations. And if a library of such specimens of sufficient variety can be collected to be shown concurrently with living cases, it will form a great aid in teaching the student to visualize the diseased conditions which come under his observation.

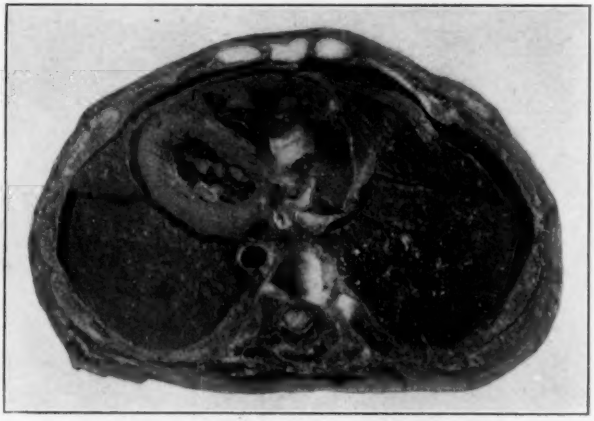

FIG. 7.—Horizontal section of child's thorax. This section shows the encroachment of the vertebral column on the intrathoracic space, the amount of space occupied posteriorly by the upper lobes, and the large area occupied by the heart.

To one who has never seen sections of the hardened thorax the obviousness of the essential facts of anatomico-clinical importance is most instructive. Among them may be enumerated the anterior, superior, posterior, and mediastinal relations of the pulmonary apices; the apposition of the right upper lobe to the trachea; the inclination to the right of the trachea; the almost directly backward course of the aortic arch; the proximity of the right base to the liver on the one hand, and of the left base to the stomach on the other; the amount of costophrenic pleural space unoccupied by the lung. In addition, there can be shown the tremendous encroachment of the vertebral column on the intra-thoracic space; the thickness of the muscles overlying the spine (and incidentally indicating the limitations of spinal percussion); the distance of the

bronchial lymph nodes from both the anterior and posterior aspects of the thorax. Innumerable points of interest are shown in regard to the heart, for instance, the posterior position of the left auricle, in relation to all the other cavities, even including the right auricle; the distance from the surface of the various valves; the relation of the aortic valve to the infundibulum, etc.

The preparation of the specimens is a comparatively simple matter. The bodies are given an arterial injection of 15 per cent. formaldehyde solution, and put in cold storage until they are frozen solid. They are then cut with an ordinary carpenter's hand-saw. As regards the plane of sections, we have been guided by the following principles: For study of the lungs, sagittal sections are the best; for the air passages, a frontal section in the axis of the trachea is of most value; to demonstrate the relations of the pulmonary bases, the frontal plane is of greatest usefulness; to show the heart to best advantage, sagittal sections are given the preference; for the superior mediastinum, horizontal and sagittal sections are of about equal value.

We have also used ordinary dissections of these hardened bodies. They are not difficult to handle, and they have the great advantage of not collapsing and of retaining their shape indefinitely.

THE TREATMENT OF PULMONARY TUBERCULOSIS BY COMPRESSION OF THE LUNG.

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Since 1825 the desirability of restricting the expansion of the tuberculous lung, and of approximating the walls of cavities, has been strongly felt by many of the best minds in the profession, and vague, inconsequent efforts have been made to accomplish these results. Carson,[1] of Edinburgh, suggested puncturing the costal pleura in order to transmit atmospheric pressure and so compress tuberculous cavities. Piorry[2] tried tightly strapping the thorax and putting a heavy weight over the cavity in order to keep the chest wall quiet, and maintained that he had seen marked improvement follow. Many distinguished clinicians have reported unexpected recoveries in pulmonary tuberculosis after the occurrence of a pleural effusion, or a pneumothorax. *A priori*, it is easy to understand how anything occupying space in the pleural cavity, will proportionately reduce the size of the lung, and cripple its

[1] Canstatt: Pathologie und Therapie, Erlangen-Enke, 1843. [2] Ibid.

power to expand. Today it is evident that by filling the pleural cavity with gas, the lung may be reduced to its smallest possible compass, the walls of ulcers and cavities closely approximated, and held so, almost as immovable as in a vise.

This simple, almost selfevident, fundamental principle escaped the attention of the medical profession until 1882, when it was crystallized out of the mass of vague, generally felt, impressions, by Forlanini,[3] of Pavia, who suggested filling the pleural cavity with a gas in order to immobilize the lung and prevent its expansion. In 1894, Forlanini[4] reported a case successfully treated by this method. In 1898, Murphy,[5] influenced by the desire to defunctionalize the lung, independently conceived the idea of compressing it by filling the pleural cavity with nitrogen, and published a report of 5 cases treated by this method. The next year, 1899, his assistant, Lemke,[6] reported 53 cases. Unfortunately Lemke died, and Murphy, although repeatedly urged to use the method, and fully convinced of its scientific value, persistently refused to do so because it would take him out of surgery into medicine. In 1905, 1906, and 1907, Forlanini's[7] reports attracted the attention of Brauer, now Chief of the great Eppendorfer Institute in Hamburg. This eminent investigator became thoroughly convinced of the value of the method and soon Lucius Spengler, and Ludwig von Muralt, of Davos, were associated with him in its use. They were followed by some of the most eminent specialists in Europe, and at present some 400 cases have been reported, so that sufficient data has been accumulated to furnish material for a critical estimate of the value of compression of the lung by means of nitrogen injections into the pleural cavities in the treatment of tuberculosis. These reports show that the method is not applicable to all cases because of pleural adhesions and pleural neoplasms, but that when it can be used, and the lung is compressed completely, and kept so for a sufficient length of time, recovery has invariably followed.

As a rule, compression of the lung by means of an artificially produced pneumothorax has only been attempted in otherwise hopeless cases. It has been universally assumed that this should not be tried until all else has failed. When the whole of one lung and occasionally part of the other had become involved it was felt that the gravity of the condition justified the seriousness of the procedure. Therefore, the cases reported are practically all of a hopeless, desperate nature. Many cases recovered, others succumbed, but in all cases, failure was due, not to the use of the

[3] Gazzetta degli Ospedali, August, September, October, November, 1882.

[4] Münch. med. Woch., 1894, No. 15; Gazzetta medica di Torino, 1894, Nr. 20 e 21; 17 e 24 Maggio.

[5] Oration on Surgery, Jour. Amer. Med. Assoc., 1898.

[6] Jour. Amer. Med. Assoc., 1899.

[7] Gazzetta medica di Torino, 1895, Nr. 44; Deutsch. med. Woch., 1906, Nr. 35; Gazzetta med. Italiana, 1907, 1908.

method, but to inability to employ it properly because of complications. For example, if the lung is held fixed by dense pleural adhesions it cannot be compressed. If the pleural neoplasms are too hemorrhagic it is difficult to enter the pleural cavity. Renal or intestinal lesions may be aggravated by the pressure, and other serious complications, such as diabetes or incompetency of the heart, contraindicate attempts at compression.

If a patient with pulmonary tuberculosis is doing badly in spite of climate, nursing, and medical skill, what then remains? If, after months of absolute rest combined with every advantage and aid that can be procured, the process steadily advances, and more and more of the lung becomes involved, what shall we do? Why should we not check this extension of the disease by actively compressing the affected lung until the fluids and decomposing masses are driven out of the lung through the bronchial tubes; until the circulation is so suppressed that the lung becomes clean, firm, and dry, and a passive stasis so encourages an exuberant development of connective tissue that all the tuberculous lesions are converted into durable scars, and all signs of tuberculosis are completely obliterated?

The cases so far reported are practically of one type. The unsuccessful ones vary according to the nature of the cause of the failure. The successful ones are all much alike, and for the most part represent advanced and hopeless cases with one lung completely involved and often the apex of the other, usually with a history of years of unsuccessful attempts at recovery under the most favorable circumstances, and showing fever, night sweats, profuse, purulent, expectoration containing tubercle bacilli and elastic fibers, emaciation, and prostration.

When nitrogen is injected into the pleural cavity, and more and more pressure is obtained, the typical results are manifested in proportion to the degree of compression of the lung. At first the temperature may rise, the pulse quicken, and the amount of sputum increase because of the pressure, but when these first effects are over, the lessened production and absorption of toxins is shown by a fall in temperature and pulse rate, and diminished expectoration. With complete compression of the lung, the breath sounds disappear, or are heard only as a metallic whistling. There is a drum-like resonance over the entire side except over the shrunken lung, which the *x*-rays show has little or no ability to expand. All this is but preliminary. The enemy has merely been driven out. The real reconstructive processes must complete the work, so that recovery will be of the most durable and permanent nature, and no future relapses can occur. While the lung is compressed it is safe, for no infection can enter. It is a more difficult task to put the lung in such condition that it will not always have to be compressed, but may with entire safety to the patient re-expand and

resume its function. This is accomplished by maintaining sufficient pressure within the pleural cavity to hold the lung closely together so that the reparative processes may never be torn. Thus an opportunity is given for fibroblasts to shoot out from the walls of the injured bloodvessels, lymphatics, and bronchial tubes into the injured tissues and for cicatrizing processes to convert the walls

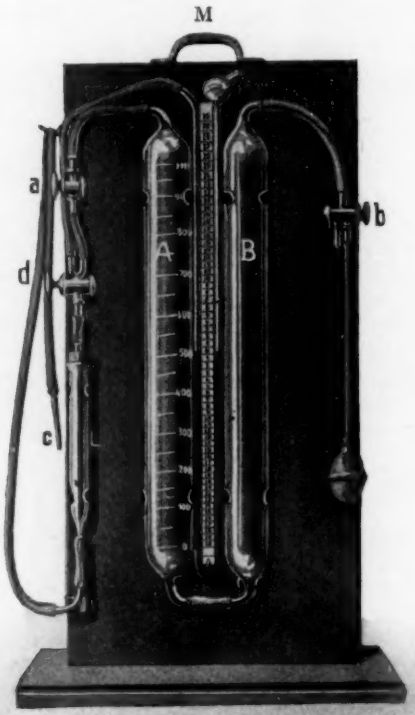

FIG. 1.—Forlanini's apparatus, modified by Saugman.

of ulcers and cavities into firm, durable scar tissue. Fortunately, this organizing invasion of fibroblasts does not concern itself with sound tissues. Elaborate experiments[8] have proved conclusively that the sound portions of the lung are in no way affected by this process. Moreover, uninjured alveoli show no tendency to adhere

[8] Brauer's Beitr. z. klin. Tuberkulose, Band xii, Heft 1; ibid., Band x, Heft 3.

no matter how long the pressure has been kept up. After a year or more when the pressure is removed the healthy alveoli readily separate and resume their function.[9]

In order to insure complete anatomical recovery, it is generally agreed that the lung must be kept firmly compressed for about a year in uncomplicated cases before it is safe to allow it to expand. At first the nitrogen is injected frequently, then less often as the pleural surfaces lose their power of absorption. In the beginning the injections are made every other day, then twice a week, and later on once or twice a month will suffice.

The simplest and most portable apparatus for making nitrogen injections into the pleural cavity is that devised by Forlanini and modified by Saugman by the addition of a manometer (see Fig. 1). The apparatus is mounted upon a board, and consists of two cylinders, *A* and *B*, each holding about a liter, and connected at the bottom by rubber tubing. Between the two cylinders is a U-shaped water manometer (*M*) 50 to 60 centimeters long. The graduated cylinder *A* contains the nitrogen; the cylinder *B* is filled with an antiseptic solution; *d* is a three-way stopcock connecting the needle-tube *c* with *A*, or with the manometer *M*. When the needle tube is connected with *M*, the manometer indicates the intrathoracic pressure, and the respiratory excursions; when connected with *A*, the nitrogen escapes into the pleural cavity. *L* is a glass filter through which the nitrogen must pass both on entering and leaving the cylinder. There is another three-way stop cock, *b*, connected with a bulb. At present this apparatus must be imported.

Brauer[10] modified Murphy's[11] apparatus by the addition of two manometers, one for water and one for mercury (see Fig. 2). This apparatus may be easily made by using a simple U-shaped water manometer with the two jars. The nitrogen jar *N* is filled with sublimate solution. The water jar *S* is lowered and the nitrogen turned on. The nitrogen is filtered through a glass tube packed with sterile cotton. The supply tube of the nitrogen jar is connected with a three-way stopcock *D* which will either permit the nitrogen to pass into the pleural cavity through the needle *P*, or transmit the interpleural pressure to the two manometers, *W* for water, and *Q* for mercury.

Before attempting to compress a lung, the functional capacity of the other lung and the effect that the increased respiratory demands will have upon it must be carefully estimated. It is not so much the extent, as it is the nature and location of the process

[9] Brauer's Beitr. z. klin. Tuberkulose, Band xii, Heft 1, S. 49 to 154; Münch. med. Woch., 1906, Nr. 7; Therapie der Gegenwart, June, 1908; Deutsch. med. Woch., 1906, Nr. 17; Ueber Pneumothorax, Programm der Feier des Rektoratswechsels, Marburg, a/L.; Wien. Therap. Woch., 1908, Nr. 33, S. 454; Therap. Woch., 1908, Nr. 29.

[10] Brauer's Beitr. z. klin. Tuberkulose, Band xv.

[11] Loc. cit.

in the second lung that will determine the advisability of attempting compression. One lung may be safely compressed when a much greater portion of the second lung is in an old, dry, cicatrized condition than if it is involved in a wet, pneumonic process, and apical lesions are not as dangerous as those situated centrally.

There are two sources of error likely to confuse us in estimating the condition of the second lung. The rales heard over it may be chiefly transmitted from the other lung and entirely disappear as soon as the latter is compressed. On the other hand, deep, central lesions, quite unsuspected at the beginning, may be so aggravated by the increased functional activity of the lung that it will be unwise to persist in the treatment.

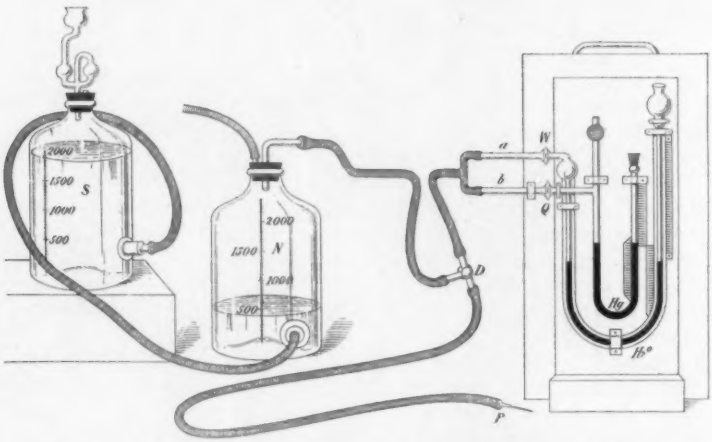

FIG. 2.—Brauer and Spengler's modification of Murphy's apparatus for nitrogen injections.

The technique of making nitrogen injections into the pleural cavity is as follows: Choose a spot for making the injection over an area where the breath sounds and resonance are best, in as wide an intercostal space as possible, avoiding the heart, the diaphragm, and the thicker muscles. Forlanini's method is to place the patient so that the selected site for injection comes uppermost, and arrange the arm so as to widen the intercostal space. Disinfect the skin with tincture of iodine and freeze with ethyl chloride. A fine hypodermoclysis needle may then be thrust through the chest wall until the pleura is felt to yield. Connect the needle with the manometer. If there are no excursions the needle is plugged or is in the lung or a bloodvessel. If for any reason the fine needle is not satisfactory, Murphy advises making a small incision just large enough to admit a medium-sized aspirating needle with a slightly

blunted point. As the pleura is felt to yield, air will be sucked through the head of the needle if there are no adhesions. If this sound is not heard the needle is either stopped up or is in the lung. The needle is then connected with the manometer, and if there are no excursions a new attempt must be made. The safest and most reliable method is that of Brauer, who makes an incision sufficiently long to afford a good inspection of the pleural surface after the tissues are retracted. If the pleura is glistening and smooth, and the motions of the lung are visible, he punctures the pleura with a blunt needle, carrying a fine catheter with which the pleural layers may be explored. When satisfied as to the existence of the pleural cavity, the needle is connected with the manometer. After the nitrogen has been injected, the incision must be closed by carefully suturing each layer of tissues. The after-fillings are made by using a fine needle without an incision.

FIG. 3.—Typical curve of pressure within the pleural cavity following nitrogen injections.

The quantity and the frequency of the injections vary with each case. When there are no adhesions and the heart action is good, larger quantities at longer intervals may be given. If the heart is weak the pressure should be kept as low as possible and the injections given just often enough to maintain the standard desired. It may not be advisable to raise the pressure much until the heart is found to be equal to the task. When the patient is not incommoded by the pressure, 7 or 8 centimeters will not be too much. Each case is a law unto itself and the proper standard of pressure must be determined for each individual, and then maintained by giving the proper quantity at the right time (see Fig. 3).

The size and situation of the lung and the degree of compression is best shown by the *x*-rays. Failing this, the character of the breath sounds indicate the density of the lung, and the area of resonance the extent of the pneumothorax. As the lung becomes compressed, the rales and breath sounds give place to metallic breathing and, finally, to a sharp, metallic clinking, or the sound of a drop falling into a metallic space. When the lung is held out by adhesions, the breath sounds and rales persist over these areas, and metallic conditions are not induced. Bulging over the

previously shrunken side is frequently seen, and the difference in motility plainly felt.

When the pleural layers are held together by bands or adhesions, the clinical course of the treatment is altered, and the technique greatly increased in difficulty, so that we encounter all degrees of failure, from inability to produce a pneumothorax, to inability to compress the lung after it is produced. The dangers attending the method are also increased. Failure of the manometer to show typical respiratory excursions may be due to the inability of the pleural layers to sufficiently separate because of adhesions too near the point of puncture. A little nitrogen may aid in separating the pleural layers and produce perfect respiratory excursions. This is tempting, but dangerous, because without the manometer we have no idea where the point of the needle may be. If we do not have the respiratory excursions to guide us, we are unable to know whether the point of the needle is in the lung, or in a bloodvessel, or whether the needle is simply stopped up. If the pleuræ are adherent they are pierced as one and the needle goes on into the lung. In all probability a fine needle will not injure the lung unless it is moved about sufficiently carelessly to tear the lung tissue. If a large needle is used and the stiletto vigorously employed to ascertain the reason of the obstruction, the lung tissue may be considerably injured and death result from the formation of an abscess. If the wall of a pulmonary vessel is torn, air may be sucked in from the lung, or nitrogen may enter. The point of the needle may be in a bloodvessel, and the nitrogen enter the circulation directly. There is always the danger of embolism whenever a filling is made. Careful technique avoids the introduction of the nitrogen into the circulation, but there are two other theoretical dangers to be considered. It is quite possible that an angiomatous condition of the pleural vessels will cause the needle to tear one of the thin vessel walls without the needle becoming obstructed. Then as the nitrogen is injected it may be forced into the torn vessel. If there are clots in the pleural or pulmonary vessels, the pressure resulting from the filling may expel one into the circulation and thence to the brain. The cerebral phenomena of embolism are loss of consciousness, rolling of the eyeballs upward, contraction of the pupils or dilatation, loss of pupillary reflex, conjugate deviation, extension convulsions, trismus, instantaneous cessation of the circulation and respiration, or prolonged failure.

Very different are the phenomena due to insult to the pleura or the pneumogastric nerve, known as pleural reflexes. These vary from slight collapse or dyspnea to complete aphonia and spasm of the glottis. They are avoided by the preliminary use of morphine, whisky, by touching the surface of the pleura with cocaine, and by not using nitrogen below the body temperature. Insult to the pneumogastric nerves may cause paralysis of coughing and speech.

A peculiar source of difficulty and danger is found in an excessive development of the pleural circulation resulting almost in a neoplastic overgrowth. Under such circumstances it is difficult to introduce the needle into the pleural cavity without the needle becoming filled with blood, and even after a pleural cavity of some size has been created, we may fail in the attempt to find it.

If too much nitrogen is introduced under too high a pressure, the nitrogen may escape back through the track of entry and appear subcutaneously as an emphysema, or it may infiltrate beneath a muscle and lift it up. Instead of leaking out, the nitrogen may produce bulging of the unsupported anterior and posterior portions of the pleura with subsequent escape into the mediastinum, forming deep emphysemas, which crowd up into the neck or press against the heart causing alarm and distress to the patient.

My own experience with the method is far too recent to be conclusive, but the results obtained are so striking that I feel warranted in reporting them. In the cases in which I have thus far employed this method of treatment the indications for the operation were: Inability to arrest the process in 15 cases; inability to hold a previous recovery in 3 cases; impatience to return to work and unwillingness to risk the uncertainty of symptomatic treatment in 2 cases; while in another patient the operation was done on purely theoretical grounds; and 1 case was referred expressly for the treatment.

There were 3 deaths and 1 failure in the 15 cases, all of which were advanced and otherwise hopeless. One death was due to hemorrhage before the effects of the method could be obtained. This case was characterized by severe and persistent hemorrhages which were the indication for attempting the treatment. The second death was due to a violent exacerbation of an intestinal tuberculosis, together with an exaggerated course of tuberculin given after the patient left Highlands. The third left Highlands far too soon and was afterward not properly cared for. The case that did not improve still has a chance of recovery. She is gaining slowly and is in every way much better than before the attempt was made. The pleural cavity is filled with adhesions and there is an enormous, almost malignant development of the pleural collateral circulation. All of the others are doing well, and in all of them recovery could not have been made unaided.

Of the 3 cases unable to hold a recovery when made, 1 has been earning his living for over a year without the loss of a day's work, and the other 2 are in equally favorable condition.

There were 2 cases of business men impatient to resume work, and unwilling to risk the slow and uncertain results of symptomatic treatment. It is too soon to prophesy, but the lungs of both are being successfully compressed, and we have every reason to believe that within a year these men will return to work and be as free from the dread of a relapse as an anatomical recovery can make them.

A recovery after the removal of all foul matter and the complete suppression of all tuberculous activity, with their lesions converted into clean, strong scars, is a vastly different matter from the shutting-in process.

The theoretical case is one in which a tuberculous process is grafted upon an emphysematous lung. The heart, overburdened with athletics, alcohol, and tobacco, and stretched far to the right of the sternum, was unable to maintain a good pulmonary circulation. The lung was stiff with blood, the thorax rigid, the patient gasping for breath, and exhausted by the profuse expectoration consequent upon the vicarious development of the bronchial circulation. Arguing that collapse of the lung is fully as necessary as expansion, and that simply the separation of the pleuræ, thus allowing the lung to fall back a little from the chest wall, would give some support toward driving the blood out of the lung, we injected 200 c.c. of air and repeated this twice within two weeks. The relief was marked and persistent, the bronchial circulation is subsiding, the rales have disappeared, the temperature, previously 99° to 101°, is normal, the breath sounds are no longer wheezing, and the patient is much more comfortable and has no dyspnea.

The referred case is that of a young woman with an old inactive process in the right lung, and an active one in the left lung involving the upper two-thirds. The circumstances at the Sanatorium not being suitable for the patient, she was referred to one of the most scientific hospitals in the East, where the lung is being compressed with marked success.

A brief summary of some of the cases treated will best illustrate the results obtained by compression of the lung with nitrogen injected into the pleural cavity.

Case I.—Schoolgirl, aged seventeen years; weight, 97 pounds. Illness of fourteen months' duration, characterized by periodic failure to menstruate and consequent exacerbation of cough, followed by vomiting and hemorrhages. The right lung was normal. The left lung showed in front, down to the fourth rib, and behind to the eighth rib, dulness and altered breath sounds. In the intervals of recovery the breath sounds were suppressed and expansion was uneven. During the failures to menstruate large and medium moist rales were heard over these areas. The sputum was full of tubercle bacilli. The indications for the operation were: Failure to hold a recovery after it was obtained; persistent hemorrhages; and inability to check the extension of the tuberculous process on account of the effects of vicarious menstruation.

The first injection of nitrogen into the pleural cavity was made on April 24, 1910, by Dr. Murphy, at the Mercy Hospital in Chicago. Nine-hundred cubic centimeters were first injected, and about the same amount was given twice within the next ten days. In the

first twenty-four hours the temperature rose to 103°, and the pulse to 120. There was a great deal of coughing and expectoration, and the patient could not lie down nor sleep. The following fillings gave little discomfort, and the return journey was much more easily made than the one to Chicago. The fillings were kept up at the Highlands Camp Sanatorium, and after two months the tubercle bacilli disappeared from the sputum. Today the patient is well and strong, has no cough, no expectoration, eats well, sleeps well, and her condition is in every way satisfactory. Since January, 1911, the fillings have been given by her home physician.

Case II.—Lawyer, aged thirty-five years. The patient had moderate expectoration with an abundance of tubercle bacilli. He had been ill for two years, and was unable to build up by eating because of violent attacks of indigestion whenever any attempt was made to force the feeding. The left lung was normal throughout. The right lung did not reveal by physical signs the extent of the lesions because they were deep and central. The *x*-rays showed considerable infiltration in the middle and lower part of the upper lobe. For a year he made constant recoveries which he lost at every attempt to make physical efforts. Indications for compressing the lung were: The difficulty of healing central lesions; the inability of the patient to make or hold a recovery; and the necessity of supporting his family.

In August, 1910, an injection of 500 c.c. was made according to Murphy's method. There were no difficulties, and after the fillings had been kept up for two months the lung was thoroughly compressed, the tubercle bacilli had disappeared from the sputum, and the patient was able to return home, where the fillings were given by the family physician. In spite of two severe attacks of grippe, the patient has not lost a day's work, and has supported his family ever since. He is today well and stronger than ever before. He has kept up the fillings until the present time, but has now discontinued them.

Case III.—A young, married woman, extremely frail, and almost unable to eat, who complained of an harassing cough, insomnia, and profuse expectoration loaded with tubercle bacilli. The patient had been ill for three years, and was unable to arrest the the process. The indications for the operation were: The profound cachexia, the failure of three year's symptomatic and tuberculin treatment, and the condition of the left lung, which was completely involved and riddled with destructive foci. In August the lung was compressed by Murphy's method without any difficulty, and by October the patient was riding, driving, picnicking, eating, and sleeping well. In December the patient returned home, the fillings were given, but an unwise mode of life converted what might have been a brilliant recovery into failure and death.

CASE IV.—Married woman, aged forty years, who had an harassing cough, and abundant expectoration loaded with tubercle bacilli. Eighteen months' rest in bed in the open air had failed to arrest the process or check the persistent hemorrhages. Over the entire right lung resonance and breath sounds were normal; over all the left lung there was dulness and the breath sounds were altered. At the level of the second left intercostal space, about 3 cm. from the sternum, there was a cavity. Indications for the operation were: Inability to check the process or the hemorrhages, and eighteen months of ineffectual efforts to recover.

In November, 1910, we cautiously began compressing the lung, fearing to burst the thin walls of the cavity. Using Murphy's method we succeeded without any difficulty, and in December of the same year the tubercle bacilli disappeared from the sputum. In May, 1911, the fillings were discontinued, and the lung allowed to expand. Today there is no cough, no sputum, and over the left apex there is dulness and irregular expansion, but over all the rest of the lung, normal resonance, and vesicular breath sounds are present.

CASE V.—Actor, aged twenty-six years, of frail, delicate physique. There were quantities of tubercle bacilli in his sputum. His history was that a few months previously there had been a violently acute onslaught of the disease, with high temperature, quick pulse, and repeated hemorrhages. This was a case of a deep, central lesion not well shown by physical signs. Normal resonance and breath sounds were present over the entire right lung. Over the left lung, to the fourth interspace in front and eighth rib behind, expansion was uneven, and there were numerous rales. The operation was indicated because of inability to keep down the temperature below 101°, or the pulse below 110, when the slightest physical efforts were made. After four months of inability to recover and the occurrence of a hemorrhage we concluded that the results of compressing the lung were more definite and certain in their effects than further waiting.

On November 1, 1910, the first filling was made according to Murphy's method. Since January, 1911, there has been no fever, no sputum, and the pulse has been 84. The tubercle bacilli have been persistently absent. Since April, 1911, no more fillings have been given, the lung has re-expanded, and resumed its function. Over all the lung there is now normal resonance and breath sounds.

CASE VI.—Woman, aged twenty-four years, frail and badly nourished, with an harassing cough, an abundance of tubercle bacilli in the sputum, and a temperature of 101° to 102°. For two years she had made ineffectual efforts in Colorado and elsewhere to recover. Over all the left lung there was dulness and there were somewhat suppressed uneven breath sounds and rales. The operation was indicated, since the future offered little more hope for

recovery than the past, and the harassing cough, anorexia, a rectal fistula, casts, and albumin were not encouraging.

In November, 1910, the first filling was made according to Murphy's method. Pleural adhesions, and the inability to endure enough pressure to break them up made the complete compression of the lung impossible. Nevertheless, the patient has of late only had fever during attacks of bronchitis in the right lung. Today she is in much better condition than a year ago. The breath sounds over the greater portion of the affected lung are suppressed, the casts and albumin have disappeared, and the patient is steadily improving. When this is contrasted with her former condition we have every reason to be gratified by the result.

CASE VII.—Actress, aged twenty-four years, fairly well nourished. The upper lobe of the left lung, and the upper and middle lobes of the right lung were involved. In the second intercostal space to the right loud, bronchial breathing and dropping rales were heard. Her temperature was 101°. She had a violent cough, profuse expectoration, and night sweats.

We hoped that the temperature was due to secondary infection of the cavity and that approximation of its walls would do more than anything else to benefit the patient. On November 1, 1910, the first filling was made. The results were most gratifying. The temperature fell to normal, appetite and strength improved. In April, 1911, the patient was able to ride and we began to hope that we could eventually allow the lung to reëxpand, and if necessary, compress the left lung. Unfortunately, the patient, tired of restraint, left the sanatorium.

CASE VIII.—Young, married woman. She was profoundly emaciated, had no appetite, and suffered from attacks of indigestion and high fever. The left lung was normal, but over the right lung there was absolute dulness down to the angle of the scapula, and complete suppression of breath sounds; above the angle there was harsh bronchial breathing; and over all the rest of the lung the breath sounds were suppressed and there were rales. This being an interstitial case, and trans-bronchial drainage failing us, treatment by compression seemed contraindicated; nevertheless, as recovery was otherwise impossible, we decided to make the attempt.

The first filling was made, according to Murphy's technique, without difficulty. On account of pleural adhesions it was impossible to compress the lung and pleural reflexes and pleural neoplasms made the attempts very difficult. After three months she had gained seven pounds in weight, was much stronger and better in every way. The temperature always rose after each filling, frequently as high as 104°, but there was no expectoration. The patient is better than a year ago, but we have given up the fillings, and do not expect a favorable result.

CASE IX.—Man, aged twenty-six years. He had been ill a year, with night sweats and severe diarrhea. Over the right lung dulness extended to the fourth rib in front, and the seventh rib behind, with altered breath sounds and rales. His temperature was 101° to 102°. The lung was compressed without difficulty, but the pressure had such a bad effect upon the gastro-intestinal conditions that it seemed useless to continue the attempts. The patient died in the winter of 1911.

CASE X.—Just after the attempt to compress the lung the patient had a severe hemorrhage, and died from aspiration pneumonia.

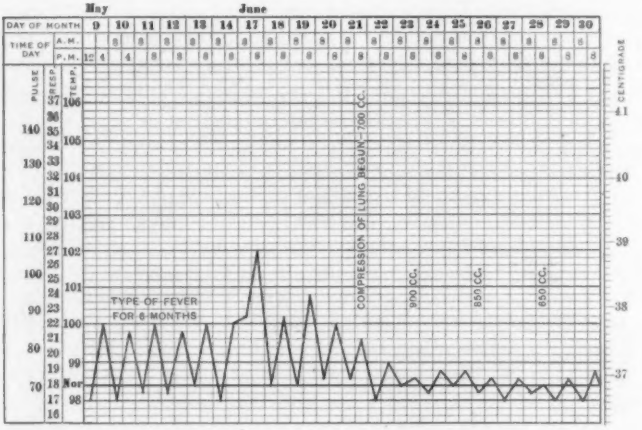

FIG. 4.—Chart showing favorable effect on temperature of compression of the lung.

Since June, 1911, we have had 14 cases, and while the results are too recent to be of any value, 6 of them are sufficiently interesting to report. Three of these cases showed a prompt and persistent fall in temperature to normal, save in 1 case during a short attack of bronchitis in the other lung. One of these cases had previously had skilled nursing for six months, with absolute rest in bed. His chart shows graphically the results of compression of the lung (see Fig. 4).

Another case was of the acute pneumonic type, with the bacilli in clumps so that it was impossible to distinguish the individual ones. There was rapid extension of the process for four months, with fever, inability to eat, and an exasperating cough. On May 27, 1911, the first filling was made. As soon as the lung began to be compressed the temperature fell to normal and has remained so

ever since. The tubercle bacilli are scattered singly through the field, the patient is feeling well, and keeping house.

In another case, rest in bed for weeks failed to reduce the temperature, but as soon as the lung was compressed the temperature became normal and has remained so.

A fourth case is that of a patient who for six months was unable to make any improvement in spite of complete rest in bed. In this case the temperature did not become normal until three months after beginning the compression of the lung.

In the fifth case the left lung was riddled with destructive foci. It was held fixed by extensive adhesions, and the pleural surfaces were covered with angiomatous vessels. Since compression of the lung was begun the breath sounds have been greatly suppressed; there are no rales, and the temperature is normal. There is a marked subjective improvement and gain in weight and strength.

The sixth case is that of complete involvement of the left lung. Instead of being a weak, bed-ridden patient, he is now feeling better than in years, and is independent in his actions. The lung is perfectly compressed, and there is no clinical suggestion of tuberculosis.

SUMMARY. There are hopeless cases of pulmonary tuberculosis which cannot recover with ordinary methods of treatment under the most favorable circumstances. There are also cases that are unable to hold a recovery after it has been made. In these cases, if the lung can be compressed, and kept so for a sufficient length of time, recovery will usually follow. Fifty per cent. of advanced cases treated by compression have been reported as permanently cured.

The durable nature of the recovery under this form of treatment is worth considering. Instead of the uncertain restraint of an encapsulating process, with all the dangerous factors of the disease merely rendered latent, we have their complete and permanent removal, and as a result the lung becomes healthy and clean. The disappearance of the tubercle bacilli removes a source of infection months before it can be accomplished in any other way.

The course of recovery is helpful subjectively. The certainty and precision of the method permit a great deal of liberty and the sense of security gives buoyancy to the patient.

When should we attempt to compress a tuberculous lung and when not? If complications exist, sufficient in themselves to forbid recovery, it will avail little to compress the lung. If the sounder lung is not too much involved, it may be greatly benefited by the removal of toxins from the other lung. At present it is generally agreed that only advanced and hopeless cases are suitable for the method. Forlanini, however, urges its use in early cases. Murphy says that the method is preëminently indicated in the early cases

because they forestall practically the only obstacle to success, namely, pleural adhesions.

A lung that is not densely infiltrated and has no adhesions is easily compressed, and the results are brilliant. The dangers and difficulties increase in direct proportion to the amount of lung infiltrated and held out by pleural adhesions, to the degree of obliteration of the pleural cavity, to the distention of the pleural vessels, and to the amount of clotting that has occurred in the pulmonary vessels.

A conservative plan is to give the patient every opportunity to recover by the usual methods provided always there is no danger in waiting. If, after persistent efforts, the temperature cannot be reduced, nor the extension of the process checked; if, after making a good recovery the patient is unable to hold it, but loses whenever any physical effort is made; or if there is some complicating factor, like vicious menstruation that predestines failure; then an attempt to aid the patient by compressing the lung seems amply justified.

LEUKEMIC TUMORS OF THE BREAST MISTAKEN FOR LYMPHOSARCOMA.

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The case herewith reported is so remarkable that it deserves record. So far as we know it is unique, and from it one may learn several lessons. The history is as follows: A married woman, aged thirty-three years, was admitted to the hospital, June 13, 1911. She said that two weeks previously she had noticed a lump in the right breast which was not tender nor painful. It had not grown any since she noticed it. There was no loss of weight. She had one child, now aged eight years. She never had had any inflammation of the affected breast. Examination showed in the right mammary gland at its upper and outer quadrant a small nodule, the size of a hickory nut, not tender, nor cystic, firm to the feel, and not attached to the skin. No axillary nodes were palpable. The remainder of the breast contained no nodules nor were there any in the opposite breast.

On June 14, 1911, the tumor was excised. A frozen section at the operating table was not satisfactory so the wound was closed. A subsequent pathological examination showed the tumor to be

benign, the diagnosis being a lymphoma. The patient went home. She returned to the hospital five months later saying that after she had been home a month another small lump developed near the old scar in the right breast and this was followed by the appearance of two or three other lumps in the same breast. Two weeks prior to her second admission into the hospital, she noticed a lump in the left breast. All the tumors had been growing steadily larger since she first noticed them. She had had no pain in them and they were not tender. There was no discharge from the nipple nor was there any loss of flesh or strength.

In the right breast beneath the nipple were three hard, nodular masses, movable, circumscribed, not attached to the skin which was movable over the tumors. In the right axilla there was felt a mass the size of a hickory nut. In the left breast in its upper quadrant were two distinct masses, freely movable and hard. Nothing was felt in the left axilla. All the other organs were normal. The spleen was not felt. There were no other tumors anywhere, no enlargements of any of the lymphatics, except in the right axilla. The Wassermann reaction was negative. The patient was seen by Dr. Joseph A. Blake, who expressed the opinion that radical operations should be done on the breasts, since the tumors were probably malignant (sarcoma), although the outlook for a permanent cure was very gloomy. It was unfortunate that no blood examination was made at this time. Had a differential count been taken the true condition, leukemia, would have been evident and the patient spared unnecessary operations. She was examined by a number of men not one of whom suggested the advisability of a blood examination. To avoid such mistakes we would suggest in all institutions where it is possible, that a routine differential blood count be taken in all surgical cases.

On November 7, 1911, the radical removal of the right breast with both pectorals and the contents of the axilla was performed by one of us (McWilliams). Numerous enlarged glands were removed, one being from the very apex of the axilla. Perfect and undisturbed primary union followed this operation.

Pathological Examination. The nipple was not retracted but appeared normal. Throughout the breast substance particularly just above and outside the nipple were several rounded masses varying in size from a pea to a hen's egg. Between these larger masses could be seen, on closer inspection, small circumscribed lobules 3 to 4 mm. in diameter, in places closely packed together, in others arranged as strands and then sheets. These smaller masses corresponded in some instances to normal breast parenchyma, in others they were fringed about the large nodules. They differed from the ordinary glandular appearance of normal breast parenchyma in that they stood out from the stroma and showed their lobulated character very distinctly. The substance of the larger

and smaller nodules appeared the same. They were of a rather dull or grayish-pink color, firm and fleshy though not hard, quite even and homogeneous, showing no areas of necrosis nor hemorrhage nor trabeculæ of any sort. The edges of the nodules were circumscribed, but showed no capsule and were seen to be attached to the surrounding subcutaneous tissues as an evenly outlined infiltration. The skin was nowhere attached to these nodules, but some of them were intimately connected with the fascia covering the muscle. There were many soft lymphatic nodes in the axilla varying from 0.5 cm. in diameter to 2.5 cm. They appeared evenly covered, dull pink, and homogeneous in substance. The muscles showed nothing abnormal.

Microscopic Examination. The nodules showed an enormous number of small cells with scarcely visible cytoplasm consisting chiefly of a nucleus, that is, round or ovoid, clear-staining, but containing particles of chromatin that often stained very deeply. These cells were supported by a delicate stroma clearly seen by Mallory's stain. There were few bloodvessels in the area, but where the cells were near vessels, the vessel wall was not disturbed. In less dense areas where in the gross small lobules could be made out, these cells were found to closely surround the acini and terminal ducts of the breast lobules and were chiefly confined to the intralobular stroma. No extensions into the muscle substance were found. At one point in the axillary tissues a vein was found containing a number of small, round cells which might have been lymphocytes or tumor cells from their appearance. The normal anatomy of the larger lymph nodes was somewhat lost. They showed no germinal centres and were made up of small, round, mononuclear cells resembling lymphoid cells. They showed chronic hyperplastic lymphadenitis. The diagnosis of lymphosarcoma was made.

With this apparently clear cut diagnosis, operation was performed on the left breast on November 15, 1911. The radical operation for removal of the breast with both pectorals and the axillary contents was performed. Considerable shock followed this second operation, and that evening the patient's condition was poor with a weak, irregular pulse, and considerable dyspnea. There was very little response to two saline intravenous infusions, so blood transfusion was performed at 9 P.M, her husband being used as donor without preliminary testing of the hemolytic action of the two bloods. Immediately before the transfusion the patient's red cells were 3,000,000, and immediately after it they numbered 4,000,000. Elsberg's cannula was used with good result. The next day the patient's condition was perfectly good, her pulse being strong and steady. There was likewise perfect and undisturbed primary union of the wound on this side. Pathological examination of the left breast showed conditions precisely similar

to those of the right breast with the exception that the nodules were considerably smaller and more isolated, while the lobular appearance of the breast tissue showed more distinctly.

One week later, at the suggestion of the pathologist, a differential blood count was made, whereupon the diagnosis was cleared up. It showed 117,000 white cells of which 90 per cent. were lymphocytes. The successive blood counts were as follows: November 22, leukocytes, 117,000; polymorphonuclears, 8.4 per cent.; lymphocytes, 90 per cent. November 27, leukocytes, 180,000; polymorphonuclears, 2.6 per cent.; lymphocytes, 96 per cent.; red blood cells, 3,000,000; hemoglobin, 65 per cent. December 2, leukocytes, 280,000; polymorphonuclears, 2.25 per cent.; lymphocytes, 96 per cent.; hemoglobin, 30 per cent. December 4, leukocytes, 250,000; polymorphonuclears, 1 per cent.; lymphocytes, 96.5 per cent.; red blood cells, 1,600,000; hemoblobin, 25 per cent.

On November 23 the patient began to have a temperature which became continuously higher each day until her death on December 14. On December 3 the examination of the eye grounds showed that in the left retina there were two hemorrhagic spots on the temporal half near the region of the macula. The right eye was normal. She was then transferred to the medical division, service of Dr. Janeway. On December 5, the edge of the spleen was felt for the first time on deep inspiration three-quarters of an inch below the costal margin and the gums of the lower jaw were swollen, spongy, tender and showed some small ulcerations, and the left tonsil was ulcerated. On December 7, she began to have persistent diarrhea, which continued until her death.

The report of the autopsy, performed by one of us (Hanes) at 7 P.M., on December 14, 1911, is as follows:

The subject is a small, emaciated female, measuring 160 cm. Rigor mortis not present. Both breasts have been removed by surgical operation, leaving normal scars. In the posterior triangle of the neck on both sides small, bean-sized glands are felt; the glands elsewhere are not enlarged. The mucous membranes are extremely pale. On incision the panniculus adiposus over the abdomen measures about 1 cm. The abdominal cavity is free from fluid and the peritoneum is everywhere glistening. The appendix is normal. All of the abdominal organs are free from adhesions, excepting the spleen, which is adherent to the lateral peritoneum over its lower third by old dense adhesions. Both lungs collapse upon opening the thorax and there is no fluid in either pleural cavity. The right lung is free from adhesions but over the posterior surface of the left lower lobe there are old adhesions. The pericardium contains about 50 c.c. of blood-stained fluid.

Heart. The heart is of normal size. Along the course of the anterior coronary artery there are hemorrhages under the pericardium, and along the lower half of the left coronary similar

hemorrhages are seen. The valves are normal and competent throughout the heart. The heart muscle shows no abnormality.

Lungs. Left: There are tags of old adhesions along the posterior surface of the left lower lobe, and for a space the size of a dollar on the diaphragmatic surface of the left lower lobe there are several small, yellow accumulations which can be torn from the underlying pleura and seem to be fibrinopurulent in character. The lung parenchyma seems normal on section. Right: The right lung is normal.

Trachea and Bronchi. The mucous membrane of the trachea and bronchi is very pale, otherwise normal.

Spleen. The spleen is enlarged, weighs 135 grams, measures 12 x 6.5 x 3.5 cm. It is adherent along its lower pole to the peritoneum, but can be separated from it without tearing the capsule. The spleen is moderately firm in consistence, the edge is rounded. On section the spleen maintains its shape. The splenic pulp is of a greyish red color, moderately firm, it scrapes off readily with the knife. The trabeculæ are easily seen. Malpighian bodies are very indistinctly seen. There are small hemorrhages scattered through the substance of the spleen.

Liver. The liver weighs 1510 grams, measures 27 x 22 x 6 cm. The surface is smooth. The color is yellowish-red and finely mottled over its entire surface. These fine mottlings are from the size of a small pin-head to a match-head, and they do not project beyond the surface. On section the liver has about the appearance as seen through the capsule. The lobulations are not distinct. There are many small, whitish-yellow nodules scattered through the liver which range in size from a pin-head to a match-head, occasionally they are as large as a pea.

Gall-bladder. The gall-bladder and bile ducts are normal.

Esophagus. The esophagus is normal.

Stomach. The stomach shows no abnormality besides pallor of mucous membrane.

Intestines. The entire length of the small and large intestines is the seat of inflammatory changes in the mucosa which become more intense as one proceeds from the stomach. In the duodenum the mucous membrane is injected and red, and as one proceeds into the ileum this condition becomes more marked, and the mucous membrane is greatly thickened. Peyer's patches throughout the ileum are enlarged and in places slightly ulcerated. The lower ileum shows quite marked congestion and edema of its mucous membrane, and the crest of the folds are covered by a brownish, necrotic substance. The bloodvessels of the surface are markedly injected. As one passes the ileocecal valve, the congestion and edema of the mucosa is much more marked, and the crest of the folds of the mucous membrane shows a brownish, necrotic substance partially replacing the mucous membrane. The

same condition extends to the rectum where it is rather less marked than elsewhere in the large intestines.

Lymph Glands. The glands throughout the mesentery are enlarged from the size of a pea to a lima bean. Some are red and congested on section, while others show a pale yellow, succulent, homogeneous surface. The glands nowhere show necrosis. The retroperironeal lymph glands from the diaphragm to the coccyx are enlarged. They form two parallel chains, each about 2 cm. in thickness. These chains are composed of glands which, on section, present a mottled yellowish-red surface in which no normal lymph gland structure can be seen.

The mediastinal lymph glands are not markedly enlarged. The bronchial glands, however, show moderate enlargement. At the bifurcation of the trachea there is a gland measuring 3 x 1.5 cm. On section part of the gland is anthracotic, but one gets the impression that this part represents the old gland while the remainder of the gland, which is pale-yellow in color, represents the hyperplastic glandular tissue.

Pancreas. The pancreas is extremely pale, its consistence is decidedly firm. On section the pancreas cuts with an elastic feel. The pancreatic duct is patent.

Tonsils. The tonsils are enlarged to the size of a hickory nut, and both, especially the left, show marked ulceration, its surface being covered with a brownish, necrotic mass. This necrosis has extended to the surface of the left anterior pillar of the fauces.

Larynx. The larynx is normal.

Thyroid. The thyroid is normal.

There are a few small glands along the course of the internal jugular vein which nowhere exceed the size of a small bean.

Genito-urinary Tract. Both kidneys are alike. They are of normal size. The capsule strips easily leaving a very pale surface. The stellate veins stand out prominently. On section the cortex stands out sharply from the medulla because of its pallor, which is marked. The cortex measures 6 mm., the striations are faintly seen. There are no nodular accumulations seen. Both ureters are moderately dilated but free from constrictions. The bladder is normal. The uterus and appendages are quite normal in appearance.

Bone Marrow from Femur. The marrow is not increased in amount, is soft and of a dark red color.

MICROSCOPIC NOTES. *Bone Marrow.* The normal structure of the bone marrow is well preserved, but its substance is occupied almost completely by one type of cell. This cell is a mononuclear with very scanty cytoplasm. The nucleus is vesicular, rather clear and measures on the average from 6 to 8 micra in diameter. An occasional megakaryocyte is seen and there are scattered through the bone marrow small islands of erythroblasts. There are a few

cells with plentiful cytoplasm and large, round, vesicular nuclei which look like myelocytes.

Lymph Glands. Throughout the body they present the same histological appearance, which will be illustrated by a description of a retroperitoneal lymph gland. The gross structure of the lymph gland is recognizable and the capsule is intact. Here, just as in the bone marrow, one type of cell largely predominates, namely, a mononuclear cell measuring on the average from 6 to 8 micra, the nucleus being vesicular and clear and the cytoplasm scanty. Small mononuclear cells with pyknotic nuclei are occasionally seen and there is an occasional normoblast.

Thymus contains a great deal of dense fibrous tissue, within the meshes of which are masses of mononuclear cells similar to those described in the lymph glands and bone marrow. No remains of thymic tissue are seen.

Spleen. The Malpighian bodies are present. They are composed of vesicular mononuclear cells similar to those described elsewhere. The splenic pulp contains a diminished quantity of red blood cells, its substance being largely composed of vesicular mononuclear cells with here and there typical mononuclear leukocytes. There are, in addition, large mononuclear cells with rather plentiful protoplasm, the nucleus being vesicular.

Liver. In the peripheral connective tissue there are occasional accumulations of vesicular mononuclear cells with an occasional normoblast and a few lymphocytes. There is a moderate grade of fatty infiltration of the parenchyma.

Kidney. In the kidney there is a patch of leukocytic infiltration, the cells here being of the same type as those described in the lymph glands and bone marrow.

Intestines. Both the small and large intestines show diphtheritic enteritis. The mucous membrane is desquamated over considerable areas. The bloodvessels of the submucosa are greatly dilated and there is a profuse inflammatory exudate which is composed almost entirely of lymphocytes, the prevailing lymphocyte being here a mononuclear similar to those described in the lymph glands and elsewhere. There are, in addition, many plasma cells.

A section of one of the breast nodules noted upon the second admission shows the following picture: There are alveolar structures lined by cuboidal epithelium with a distinct basement membrane, which are surrounded and embedded in a mass of mononuclear leukocytes. These leukocytes measure on the average from 6 to 8 micra, and the nucleus is round, clear, and vesicular. They are obviously the same type of lymphocyte as those previously described in the other organs.

To summarize, then, the case is surely one of lymphatic leukemia, with, however, very unusual features. The patient complained first of a tumor of the breast, and probably referred any slight

evidences of impaired health to this tumor, whereas the subsequent course of the disease and the autopsy show that the breast tumor was merely an incident in the leukemic process. The superficial lymph nodes were never enlarged. The spleen was not palpable until late in the course of the disease. A study of the various blood-forming tissues reveals a transformation from the normal to a tissue in which one type of mononuclear cell greatly predominates.

The patient developed toward the close a severe enteritis, and it is interesting that the type of cell which took part in the inflammatory exudate was the mononuclear cell seen elsewhere so abundantly, and not the polymorphonuclear leukocyte, a finding easily explained by the condition of the bone marrow, in which very few myelocytes were found.

OSTEITIS DEFORMANS, PAGET'S DISEASE, WITH REPORTS OF TWO CASES AND AUTOPSY IN ONE.[1]

By B. Franklin Stahl, B.S., M.D.,

Clinical Professor of Medicine, Woman's Medical College of Pennsylvania; Associate in Medicine, University of Pennsylvania; Visiting Physician to the Philadelphia General Hospital and St. Agnes' Hospital.

The occurrence of osteitis deformans is of sufficient rarity and clinical interest to make desirable the following report of a case which came under my care in the wards of the Philadelphia General Hospital. Incidentally it may be mentioned that one of the most complete and scientific studies of this disease was made upon a patient in that hospital and reported by Drs. Packard, Steel, and Kirkbride.[2]

Historical. Paget was not the first to describe cases of the disease, for he acknowledges the previous cases of Wilks, Rullier, and Wrany. Czerny had in 1873 described a case of spontaneous curvature of the long bones of the lower limbs, to which he gave this name. In 1876 Paget[3] published an article which has given the disease a recognized place in our literature. In this paper he reported 5 cases that conformed to his classification. He subsequently reported two additional cases.[4] It seems unfortunate that the affection should be called Paget's disease, because of the confusion resulting from this name being more generally associated with a peculiar form of cancer of the breast. It would seem more desirable to call it Paget's osteitis deformans, or, better still, to omit Paget's name.

[1] Read before the Section on General Medicine of the College of Physicians of Philadelphia, December 19, 1911.

[2] Amer. Jour. Med. Sci., 1901.

[3] Trans. Path. Soc., London, 1877.

[4] Medico-Chirurgical Trans., 1882.

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Butlin and Stegmann have reported a case showing the changes of osteitis deformans in the bones of prehistoric man. That the disease is not confined to man is evidenced in the report of its existence in the bones of lower animals.

Classification. The disease is to be differentiated from osteomalacia, rickets, leontiasis ossea, leuitic osteitis, osteogenesis imperfecta, tuberculous osteitis, achondroplasia, and general hyperostosis.

Many cases in the literature do not clearly belong to this category. The article by Packard, Steel, and Kirkbride reviewing all the reported cases up to 1901 eliminated all but 67 cases. Clopton,[5] in 1906, states that the cases reported number 75.

Symptoms. Little of a definite character has been added to change Paget's description of the disease. "Most characteristic are the stooping posture, round shoulders, with head forward and chin raised as if to clear the sternum, loss of height, chest sunken toward the pelvis, pendulous abdomen, legs curved and held apart, knees slightly bent, the ankles overhung by the legs and the toes turned out. The enlarged cranium, square-looking or bossed, may add distinctiveness to these characters and they are completed in the slow, awkward gait and in the shallow costal breathing, which is compensated by the wide movements of the diaphragm and abdominal wall and the marked uplifting of the shoulders."

Subjective symptoms referable to the disease are vague and consist mostly of "rheumatic pains" in the parts affected. The insidious onset makes it difficult to determine the time of the first appearance of the disease. Paget states that it begins in middle life or later. Exceptions to this rule are evidenced in the cases of Sonneberg, of a girl, aged sixteen years; Maizard, a case, aged twenty-one years; and White, a man who had the disease when aged thirty-five years.

Parts Affected. The cranium and long bones of the lower extremities are the chief points of involvement. The lesions are usually symmetrical, but crossed involvement is often noted, *i. e.*, left leg and right arm. The left leg is most frequently first affected. The bones enlarge and soften. The location of the curvature seems to be determined by the superimposed weight, as is suggested by the spinal curvature with bowing and twisting of the femurs and bones of the legs. It has been pointed out that the deformity may be due to pathological increase in the length of the curved side of the bone. Neither of these explanations covers the cranial deformity, the patellar nodes, nor the exostoses on the arms and clavicles. The thorax frequently shows a resemblance to the rachitic type, the spine is curved and the vertebræ are fixed. The pelvis is broadened so that in the male it resembles the female type. The cranium is large, oval-shaped, with the broadest end up, and presents

[5] Interstate Medical Journal, 1906.

inequalities in the surface. The arms seem disproprotionately near the ground. The affected bones present localized nodes and circular thickenings. In some cases there were circumscribed painful swellings of the periosteum, which later subsided. The gait is "waddling." The two features of the disease that are most apt to attract the patient's attention are the increased size of the head in the male, noted because of the difficulty in procuring a hat to fit, and the loss of stature in the female, made evident by the necessity of shortening the skirts.

Etiology. Among the suggested causes are syphilis, cancer, arteriosclerosis, nerve lesions, including those of trophic origin, gout, and perversions of the secretions of the ductless glands. Syphilis of the hereditary type offers more probabilities of causation than does the acquired variety. Cancer is more often a terminal than an early condition. The evidence of any of the other suggested causes is inconclusive. It is safe to say that the cause of this disease is undetermined.

Heredity certainly exercises an influence. Chaufford[6] reports the disease in mother and daughter; Berger,[7] in mother and son; Smith,[8] in father and son; Lunn, in two brothers; Pic, in two members of a family; Robinson, in two members of a family; Oettinger and Lafont, in father and two sons; Kilmer, in sister and brother. The sister of the case here reported shows the disease in a definite but incompletely developed stage.

Pathological Anatomy. The skull and long bones are most affected, but the spinal column, clavicles and patellæ are usually involved. Symmetrical parts are not always affected. The skull is greatly thickened, the sutures obliterated, and the Haversian canals effaced. The new bone is deposited on the outer table. There is no encroachment upon the capacity of the skull. In the early stages the calvarium may be cut with a knife or compressed between the fingers. It is very vascular and later undergoes defective ossification, characterized by deficiency of lime salts. The bones of the face are infrequently involved. The new bone is not deposited in the same way as in rickets. In osteitis deformans it is formed on the convex surface about the circumference of the bone, while in rickets it is placed upon the concave surface. The origin of this new bone, according to most writers, is periosteal. Von Recklinghausen, however, maintains that it is formed from the medulla. In the process of its formation there are successive stages of absorption and regeneration. Softness and bending of the bones are characteristic, but there are to be noted in various affected areas points of ivory-hardness. The uncalcified new bone being markedly elastic, the tendency to fracture, observed in certain other bone affections, is absent in this disease.

[6] Osler's Modern Med., 1909. [7] Ibid. [8] Ibid.

Paget regards the disease as a chronic inflammation with: (*a*) rarefying osteitis, particularly of the compact bones, (*b*) formation of new bone from the periosteum.

Termination. Insanity has marked the late stages of certain cases. It has been pointed out that cancer is not as frequent as might be expected in persons as old as are most of these patients; nor is sarcoma as common as one would think likely in a disease which would apparently make a transition reasonable. The fatal termination seems to be in no way related to the bone changes.

Complications. Arteriosclerosis and cardiac disease are common. Emphysema, bronchitis, edema, pleuritis, cancer, Bright's disease, and insanity have been noted. Changes in the cord have been reported, but the findings have not been conclusive.

Diagnosis. It seems probable that few cases are overlooked and unrecorded. The greater frequency with which these cases are found in the recent literature would suggest a more general recognition of the disease. The earliest possible diagnosis may be made by the *x*-rays. The first report of *x*-ray findings in a case of this disease was made by Gallois in Gaillard's case. In it there was thickening of the diaphysis and calcification of the arteries of the extremity. The radius and ulna were also much thickened and slightly curved. The marrow was hypertrophied and its structure was altered. The ends of the joints were deformed, but the carpi were normal. In the head the shadows of the bone were darker than those of the neighboring bones. The humeri were thickened, but the elbow-joint was fairly intact despite the distinct alteration of the ends of the bones forming it. In Daser's case there was "thickening of the bones with irregular calcium deposits in the cortex," and encroachment upon the marrow cavity. In Hochkeimer's case the shadow of the marrow cavity had entirely disappeared. Schlisinger's case showed no involvement of the epiphysis. This speaks against the view that the disease is a modified form of arthritis deformans. Sonneberg found the bones of the pelvis to present an appearance similar to that of osteomalacia.

Differential Diagnosis. In mollities ossium there is not the same thickening of the bones as is seen in this disease, and the cranium is not involved. In fragilitis ossium the deformity is that which usually attends fractures. Acromegaly differs in the shape of the head and in the involvement of the lower jaw, superorbital arches, and hands. Leontiasis ossium effects chiefly the bones of the face. Hyperostosis canii is regarded by some as a stage of osteitis deformans, but others consider it a separate disease, since it does not involve the long bones.

Treatment is of no avail, and consists in palliation of the symptoms incident to the intercurrent diseases.

The following is a partial list of the cases reported since 1906, when M. B. Clopton summarized the previously reported cases.

There is a possibility that some of these cases have been included in his estimate of the number on record.

In 1906 each of the following reported a case: Bardenheur,[9] C. Mackey,[10] A. Saunders,[11] A. Tedeschi,[12] C. Sternberg,[13] E. Modea and C. da Lana.[14]

In 1907 two cases were reported by Apert and Berwait,[15] one each by H. Schlesinger,[16] R. Waterhouse,[17] and D. Stanley.[18]

In 1908 A. Herbert,[19] Sinclair White,[20] B. Bramwell,[21] J. H. Ramsburgh,[22] K. Glaessner reported cases,[23] and J. Vergner[24] observed three.

In 1909 one case each was recorded by Klippel and Pierre Weil,[25] W. R. Hewitt,[26] Prescarlo and Bertoletti,[27] Bandy and Clermon,[28] H. Koch,[29] R. A. Hann,[30] F. Ravenna,[31] two cases by R. Manwaring White,[32] and three each by C. J. Bertlett[33] and C. von Kutscha.[34] Total 34 cases.

Report of Case. E. H., married, aged sixty-five years, admitted to the Philadelphia General Hospital August 15, 1910, complaining of shortness of breath, edema of legs, and a feeling of distress in the gastric area.

Family History. Mother died, aged ninety years, of apoplexy, father died, aged fifty-three years, of apoplexy; two brothers living, one has chronic indigestion, the other is healthy; three brothers are dead, one aged twenty-one years, of heart disease, the other two died in infancy; three sisters died in infancy; one sister is living, aged sixty-seven years, has renal and cardiac disease, and shows unmistakable evidences of osteitis deformans in the bones of the head and shortening of the legs without evident thickening of the long bones. She declines to submit to *x*-ray examination. The history states that the maternal grandfather died, aged ninety-eight years, the great grandmother, aged one-hundred and five years, and the great-aunt, aged one-hundred and eight years, the

[9] Deutsch. med. Woch., 1906, xxxii, 525.
[10] Lancet, 1906, i, 787.
[11] West London Med. Journal, 1906 xi, 27.
[12] Semano Med., Buenos Aries, 1906, xiii 993.
[13] Verband. d. deutsch. path. Gesell., 1906, 137.
[14] Margagni, Milan, 1906, xlviii, 337.
[15] Bull. et mém. d. hôp. de Paris, 1907, xxiv, 235.
[16] Mitt. d. Gesell. f. in. Med., 1907, vi, 67.
[17] Lancet, May 4, 1907.
[18] Lancet, 1907, i, 889.
[19] So. Calif. Pract., 1908, xxiii, 400.
[20] British Med. Jour., 1908, ii, 1675
[21] Trans. Medico-Chirurgical Soc., 1908, N. S., xxvii, 254.
[22] Wash. Med. Annals, 1908, vii, 179.
[23] Mitt. de Gesell. f. in. Med., Wien, 1908, vii, 98.
[24] Ann. d'ocul., Paris, 1908, cxi, 321.
[25] N. incong. de la Salpêtrière, 1909, Paris, xxii, 1 to 23.
[26] Interstate Med. Journal, 1909, xvi, 561.
[27] N. incong. de la Salpêtrière, Paris, 1909, xxii, 252 to 266.
[28] Toulouse méd., 1909, xi, 16.
[29] Verhand d deutsch. path. Gesell., 1909, 107 to 119.
[30] British Med. Jour., 1909, i, 135.
[31] N. incong. de la Salpêtrière, 1909, 524.
[32] Brit. Med. Jour., 1909, ii, 12.
[33] Yale Med. Jour., 1909, xvi, 367.
[34] Arch. f. klin. Chir. Bul., 1909, lxxxix, 758.

paternal grandmother, aged seventy-nine years, and an aunt, aged seventy-five years. There is no history of tuberculosis or malignancy.

Previous History. The patient was married at eighteen, and had one child one year later. There is no history of venereal disease or miscarriages. She had rubeola and parotitis in childhood, pneumonia at sixty-three. A tumor was removed from the left breast when she was aged twenty-three years, and it has not recurred.

Present Illness. About nine years ago she first complained of shooting pains in the legs, which sometimes would extend to the shoulders. Damp weather made them worse but they were not more severe at night. Three years later she first noted that her legs were curving and that the bones of the head were becoming "lumpy." She was unaware of any change in her spine. For three years she has complained of gastric symptoms and dyspnea, and lately of edema of the ankles.

Examination. Pale, poorly nourished, and about four feet nine inches in height, which is three inches shorter than she was eight years ago. Skin loose, cachectic appearance, muscles soft, flabby, and somewhat atrophied. In bed she persistently assumes an attitude with her legs bent under her, leaning on her elbows and her head bent forward. Forehead prominent and high, temporal and frontal bones thickened and parietal bone flattened. The head measures 65 cm. in circumference; from occiput to glabella superiorly, 39 cm.; transverse measurement between meatuses, 37 cm. Orbital cavities are overhung by the frontal bones. Beginning cataracts are the only abnormalities noted in the eyes. The chest is pigeon-breasted, the lower part of sternum depressed; the scar of the amputated breast is present; respiratory movements are shallow. Abdomen prominent and pendulous, walls are edematous, and a deep transverse furrow is noted near the free margin of the ribs. The liver is enlarged. The heart is displaced to the left, the apex beat is in the seventh interspace, the muscle tone is fairly good. There are signs of mitral regurgitation and aortic roughening. There is marked arteriosclerosis, and the pulse is of good volume and high tension. The lungs show edema at the right base. The spine shows a thoracic and lumbar scoliosis.

Arms: Biceps and triceps reflexes present on both arms, resistance to passive movement, and to movement against resistance is good in both arms, no abnormality in size and shape of bones. Legs: Marked edema extending to knees. External and anterior curvature of femurs and tibias, together with thickening and enlargement. Patient cannot bring knees together closer than 17 cm.[35] The ankles are overhung by the legs. Sensation is normal

[35] Circumference of each knee, 8 cm.; of pelvis, 92.5 cm.; intertrochanteric, 31 cm.; length of spine, 52 cm.; plantar arches normal.

in the arms and legs. Leg muscles are better preserved than are those of the arms. Knee-jerks normal. Babinski and ankle clonus absent. Resistance to passive movements good in both legs; movement against resistance is poor in both legs. Speech normal, memory poor, orientation and reasoning good. Station: Shoulders bent forward, arms hanging, head elevated. Gait: Slow, awkward, scissors gait with toes turned out.

Urine shows marked albumin, epithelial and granular casts, Bence-Jones albumin test negative. Weight 96 pounds, says it was 83 pounds five or six years ago. Blood: red blood corpuscles, 3,770,000; white, 6,200.

The autopsy was performed by Dr. A. G. Ellis, assistant pathologist to the hospital and his notes are as follows:

Clinical Diagnosis. Chronic interstitial nephritis; mitral regurgitation; chronic gastritis; osteitis deformans.

Pathological Diagnosis. Hypertrophy and dilatation of left ventricle; hypertrophy right ventricle; dilatation both auricles; chronic aortic and mitral endocarditis; fibroid myocarditis; atheroma of pulmonary artery; aorta and coronary arteries; emphysema and edema of lungs; bilateral hydrothorax; partial atelectasis of right lung; chronic nephritis (diffuse); chronic catarrhal gastritis and enteritis; red atrophy of liver; hyperostosis of bones of skull and of long bones.

Autopsy thirty-one and one-half hours after death. The body is that of a markedly deformed adult white female, 141 cm. (56.4 in) in length. Rigor mortis is present. There is pronounced edema of the lower extremities and hands and to a lesser extent of the chest and scalp. The head is enormously enlarged, the lower part of the face not sharing in this change and thus giving a triangular visage with the base uppermost. Measurements: Circumference of the head, 60 cm.; over the vertex from one ear to the other, 33 cm.; from the root of the nose to the external occipital protuberance, 40 cm.; bitemporal, 14 cm.; biparietal, 17 cm.; biaural, 15 cm.; angle to angle of jaw, 11 cm.; occipitofrontal, 40.5 cm.; occipitomental, 20 cm.

Each side of the skull shows a prominence above the ear. These extend 5 cm. both anterior and posterior to the ear, and reach an elevation of 2 cm. They are practically symmetrical. Slightly above the site of the external occipital protuberance is a rounded elevation 6 cm. in diameter. The forehead is very prominent, this prominence reaching 7 cm. posterior to the supra-orbital ridge, where it rather abruptly terminates. The nose is decidedly broadened at the extremity, and the openings of the nostrils are large. The bones of the face are not appreciably increased in size.

The chest is narrow above and broadened below. The obliquity of the ribs is excessive, increasing from above downward. The upper ones are widely separated, the second and third being 3 cm.

apart at the junction with the cartilages. Circumference of the chest just above the nipple line is 73 cm.

The arms show relatively little change. The left humerus is moderately bowed forward, this being confined to the upper half of the bone. The right is slightly more bowed than the left, especially in the upper third. The forearms show no appreciable change except a slight outward bowing of the upper portion of the right radius and ulna. The hands appear normal.

The lower extremities are decidedly bowed outward. This change in both thighs and legs causes the internal condyles of the femora to be 18 cm. apart. The curving of the legs is practically equal on the two sides. The femurs are convex anteriorly and externally, the tibias the same, though in the latter the anterior bowing is more prominent. Circumference of left knee is 32.5 cm.; of right, 33.2 cm. From the left anterior superior iliac spine to the external condyle of the femur is 37.5 cm., between the same points on the right, 38.5 cm. From the internal condyle of the femur to the internal malleolus is 33.5 cm. on each side. The distance between the anterior superior spines is 26 cm. From the anterior superior iliac spine to the symphysis pubis is 15 cm. on each side. The feet show no noteworthy deformities, though the intense edema might mask some changes.

The superficial fat of the chest and abdominal wall is pale yellow in color; the muscles are pale, waterlogged, and flabby. The skin over the abdomen and lower part of the chest anteriorly is marked by numerous slightly elevated, firm, brownish patches, 2 mm. to 1 cm. in length. These appear to be sessile papillomas.

The peritoneum contains 300 c.c. of slightly blood-tinged fluid. The transverse colon is prolapsed, nearly to the level of the umbilicus. Liver is 5 cm. below the costal border. The diaphragm is at the level of the fourth rib on the right side, of the sixth interspace on the left.

The left pleura contains 350 c.c., the right 1000 c.c. of nearly clear serum. Thymus gland is not present. The pericardium contains 75 c.c. of clear serum.

The heart, especially the left side, is greatly enlarged. The right side contains clotted blood, the left is empty. The right auricle is dilated. The wall of right ventricle is firm and thickened, measuring 0.7 cm. The endocardium shows several firm grayish patches. The left auricle is dilated. The leaflets of the mitral valve are thickened and calcareous at the base. The tendinous cords are fibroid, and in most instances calcareous, the latter change extending into the papillary muscles. The endocardium of the left ventricle is gray at points. Some of the muscle columns are entirely fibroid. Those at the apex are stretched and flattened. Wall of left ventricle, 2.2. cm. thick. The muscle is pale red and very firm. The aortic leaflets are thickened and calcific at the bases. The aorta shows numerous yellowish patches, some of which are very slightly

calcified. The coronary arteries contain patches of thickening, but are not calcific. Weight of heart, 480 grams.

Microscopically the muscle fibers are granular and there is moderate increase of the interstitial tissue.

The left lung crepitates but slightly, especially near the margins, because of distended vesicles. It is grayish in color, externally and on section, and relatively bloodless. The posterior portion contains an excessive amount of serum. The right lung is similar except that the larger part of the upper lobe is collapsed as the result of pressure by the fluid in the pleural cavity. The branches of the pulmonary artery in both organs are extensively sclerotic. Weight of left lung, 280 grams, of the right, 335 grams.

FIG. 1.—Large oval head in profile, showing enormous thickening of skull (1 inch) with porosity of the bones.

The thyroid gland is slightly more adherent than usual to the surrounding structures. It is pale red in color and very flabby. On palpation it is quite resistant, but is less firm on deep pressure than is the usual organ. Weight, 9.5 grams.

Microscopically the gland as a whole is deficient in colloid. Some areas contain the usual amount, an occasional acinus in these areas being dilated and overfilled. In the greater part of the organ the acini contain only a very small colloid content or, in the majority, are devoid of that substance. The latter acini show desquamation of the lining epithelial cells which are scattered

FIG. 2.—Another view of head in profile, showing marked thickening of bone and increased porosity.

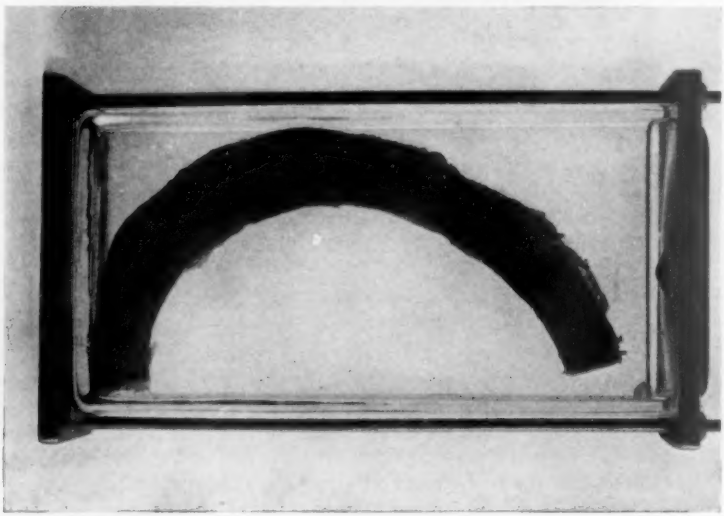

FIG. 3.—Section of skull, showing thickening of the bone (one inch), marked irregularity of the convex surface, defective ossification, and uneven distribution of compact tissue.

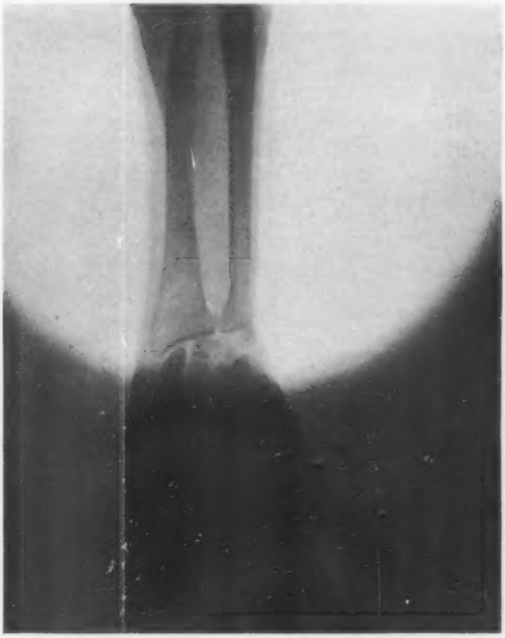

FIG. 4.—Bones of forearm, curved above and rarefied at lower ends.

FIG. 5.—Femur showing curvature, thickening of the compact tissue and encroachment upon the medullary cavity.

promiscuously through the lumina. These cells also show decided changes in the cytoplasm, consisting of granular degeneration or even disintegration, many of the cells being little more than nucleus with possibly a few adherent granules of cytoplasm. In the acini partly filled by colloid the epithelium shows almost as great a change as that just described. In most of those containing the usual amount of colloid the lining cells are still adherent to the walls, and show less prominent cytoplasmic change, although at least

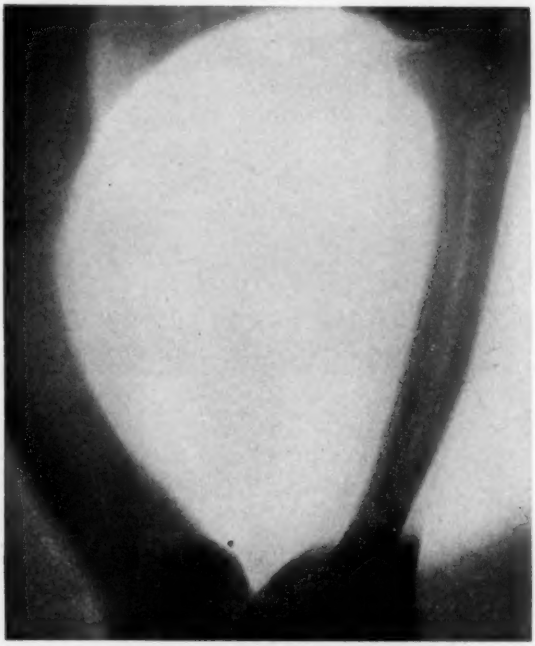

FIG. 6.—Marked outward curving of tibia and fibula with great thickening of the compact structure of shaft and irregular encroachment upon medullary cavity.

granular degeneration is present. The interstitial tissue of the gland shows no pronounced changes. Occasionally there are small circumscribed collections of mononuclear cells which encroach upon the neighboring acini. The small bloodvessels are decidedly engorged. Bodies supposed at autopsy to be parathyroid glands prove on section to be small masses of thyroid tissue.

The spleen is small, very firm, and the pulp is dark red in color. Weight, 115 grams.

The left adrenal shows no noteworthy changes. Weight, 5.5 grams.

The left kidney has a moderately adherent capsule. The underlying surface is granular. On section the organ is grey and the cortex is in many areas narrowed. The pyramids are dark bluish in color. Weight, 125 grams.

Microscopically the epithelium is granular and disintegrating, and there is considerable increase of connective tissue.

The right adrenal appears normal. Weight, 5.2 grams.

The right kidney is same as the left. Weight, 115 grams.

The ureters and bladder show no gross lesion.

The uterus contains an interstitial fibromyoma 1 cm. in diameter; otherwise it is normal. The ovaries are masses of fibrous tissue containing a few small cysts.

The gastric mucosa shows numerous dark red areas, largely postmortem in origin. The mucous membrane is not appreciably thickened.

The liver has a linear depression running the entire length of the organ, parallel to and 5 cm. above the lower border. This furrow is 2 cm. wide and 1 cm. deep. The capsule is thickened here and incision shows the fibrosis confined to that structure. The cut surface of the liver is dark red, and shows slight mottling. The organ is rich in blood. Weight, 1360 grams.

Microscopically there is an excess of blood in the central veins and adjoining cells are atrophied. The cells show granular degeneration.

The pancreas presents no noteworthy changes.

The intestines are normal. The thoracic and abdominal aorta contains many yellow patches in the intima.

The scalp over the lateral prominences already described is 2 mm. thick. Over the midportion of the skull it is 0.8 cm., but here it is very edematous. It is nowhere markedly thinned. Exposure of the skull shows on each side of the median line, posterior to the ear and extending nearly to the occipital protuberance, a dark-red area apparently due to blood beneath the pericranium. The bone saws with great ease. The skull in the median line at the anterior margin of the saw cut is 2.5 cm. thick. Posteriorly it is 1.7 cm. and at the lateral angles of the cut, 1.5 cm. thick The external table has practically disappeared as such, the bone being spongy or at points decidedly porous. For the most part this extends the entire thickness of the skull, but at points there is relatively dense bone corresponding to the internal table; this is especially true of the lower lateral portions where a diploic zone is hardly distinguishable. The upper lateral portions and the vertex show a distinct diploic zone up to 1 cm. or slightly more in width. Of the remaining denser, but still relatively soft, bone about two-thirds is internal, corresponding to the internal table,

one-third external corresponding to the external table. In these areas the diploic tissue is very porpous and vascular, and can be readily indented by the fingernail. At a few points in this zone are small islets of dense white bone, a few attaining 1 cm. in diameter.

Sections including the entire thickness of the bone were made from the lateral portion where the skull is practically uniform, diploic structure being unrecognizable. The thickness here is 2 cm. The microscopic structure is almost uniform the entire length of the sections, the extreme dural portion being only slightly more dense. In general it may be described as fibro-osteoid tissue. In all except some portions of the inner extremity the fibrous tissue slightly exceeds the osteoid in area. The latter contains no normal Haversian canals, and shows no normal lamellation, lamellæ, except in occasional areas, being entirely absent. What correspond to bone cells are numerous, but they appear immature and are in illy defined spaces.

The fibrous tissue occupies the greatly enlarged Haversian canals, and is mostly loose and cellular; in limited areas it is in the form of closely placed fibrils forming denser masses which stain deeply by Van Gieson. This tissue is everywhere very vascular. For the most part there is a sharp line of demarcation between it and the osteoid tissue, but at points where the former is densest the two gradually merge, giving the appearance of the osteoid tissue being transformed into the fibrous. The osteoid tissue is a little more prominent in what corresponds to the inner table. Sections from some blocks contain near the midpoint some quite large spaces in a matrix of mono- and polynuclear cells, marking the site of the diploe, but these areas are small.

In some sections multinuclear giant cells are quite numerous in the connective tissue, most of them being in contact with, or even nested in depressions in the osteoid tissue, resorption of the latter being in such instances very apparent. A few corpora amylacea are in the fibrous tissue. Both the pericranium and dura mater contain small islands of osteoid tissue.

The brain is pale in color, especially at the base. It is fairly firm, and shows no definite lesion. The basal vessels show only tiny scattered patches of sclerosis. Weight, 1.025 grams. The spinal cord shows no gross lesion.

Microscopically neither brain nor cord shows any noteworthy departure from the normal.

The hypophysis has a flattened anterior lobe, from above downward. The posterior lobe is very soft and was damaged in removal. The organ is not enlarged. Weight, 0.57 gram.

Microscopically the gland in general does not stain well, but there is no decided change in the anterior lobe except that all the cells are granular. Eosinophile cells are fairly numerous, but the

granules are not deeply stained. Masses of colloid in the acini are numerous. Only a fragment of the posterior lobe is attached.

Summary of the features of the case here reported: Woman of advanced years, indefinite history of onset, rheumatic pains, admitted to the hospital for symptoms in no wise related to the disease, deformity of the head, loss of stature, and swelling of the legs had been noted by the patient. These were confirmed on examination, and the bony increase detected as well as the spinal curvature. The *x*-rays and autopsy corroborated the findings.

A sister of the patient has the same disease in an early stage.

I desire to thank Dr. Walter J. Daly, resident physician, for his assistance in the preparation of the notes of the case, and Dr. Philip F. Williams for the study and report of the cultures; also Dr. Leonard Frescoln for the *x*-ray studies.

NOTE ON THE VALUE OF NITRIC ACID IN CAUTERIZING WOUNDS MADE BY RABID ANIMALS.

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The application of nitric acid to wounds made by the bites of rabid animals is something more than a conventional method of medical treatment. It offers a rational interpretation of many things in the prevention of hydrophobia that are usually ascribed to the diffuseness and loitering motion of the "virus," to that errant motion which sometimes occasions it to lose its effect or its paths of connection with the central nervous system. This paper, which is deductive in aim, is chiefly concerned with the finding of arguments that support the simple proposition that cauterization with nitric acid is in itself an immediate source of safety. The idea is to ascertain how far, by fitting a homely remedy to people in a state of probable infection, that sort of security and that quantity of security may be imparted which the physician rationally may hope to impart.

The agencies requisite for the production of the kind of security just mentioned are: (1) Hemorrhage, by which is meant the momentary and unrestrained bleeding after the bite, and (2) cauterization, that is, the destruction of the "virus" and the parts in close connection with the outlines of the wound. The more powerful and diffusible the caustic the wider will be the range of its powers, both

for purposes of destroying first and cleaning afterwards. The distinction between valid cauterization and methods less technically competent, between caustics in the strict sense and antiseptics, with which they are often confounded, will be strongly marked in the character of the caustic employed. If nitric acid be used, instead of one of the numerous antiseptics in favor, the process will resemble actual burning, with the addition of washing.

It is the mistake of much popular medicine to suppose that wounds do not heal after nitric acid. Experience at the Department of Health of New York City does not support this view. Care must be taken, however, to clean the skin, and not to apply the acid too extensively to the bloodless, bony, or cartilaginous parts of the body. It is a good practice to draw up a small quantity in a capillary pipette, and deposit it drop by drop in the wound. This avoids risk, realizes the greatest amount of effect, and facilitates measurement. It enables the physician also, from time to time, to see what progress he makes and how far he avoids any needless pain or uninfected tissue. If the wound be uncommonly severe, or the case is that of a child, chloroform anesthesia is required, a precaution that should always be borne in mind. Such is the technique of this method of cauterization which, in spite of its simplicity and excellent effect, is strangely undervalued by the profession generally, as well as by those who have some favorite hypothesis to maintain. An instance is supplied by some remarks of Remlinger,[1] still more explicitly developed by A. Marie.[2] According to the first of these writers, cauterization is ineffectual if performed an half-hour after a bite. "It cannot be affirmed too strongly," says Dr. Marie, "that no cauterization can protect from hydrophobia." If this were a true representation of the scheme of cauterization, we cannot wonder that some doctors should consider it an unnecessary attendant on vaccination against rabies. To many others the remark may well seem needlessly skeptical and greatly overdrawn.

There has been a notable increase lately in wounds made by the bites of animals. At the Department of Health we see many cases peculiarly ill-treated and neglected. Many of these come from places outside the city, but New York itself (with the advantage of newspaper mouthpieces) offers many an instance in point. It is not easy to form an accurate estimate of the mischief done; it must, however, be wholly disproportionate to the pain or trouble inflicted by the use of nitric acid. Although pain may be allayed in a great measure by the application of anodynes, since errors in the treatment of bites have a many-sided interest, it is clearly worth while to undertake a systematic defense of the true method.

[1] Bacteriotherapie, Vaccination, Serotherapie, 1909, p. 81.

[2] Kraus and Levaditi's Technik und Methodik der Immunitätsforschung, Ergänzun sband I.

The incurable optimism of doctors is responsible for two deplorable events: (1) Now that they have come to rely almost exclusively upon vaccination in suspected rabies, many have ceased to pay sufficient attention to cauterization; (2) a multitude of remedies (and theories) have acted in competition with a combined force to blunt the critical powers of the mind.

The invaluable agents of cauterization, nitric acid, the much weaker silver nitrate, and actual burning (with which the mind readily associates excision) have been driven into neglect by dubious novelties and the increasing accumulation of rival eccentricities in medicine. The controversy as to the merits or the demerits of caustics is worldwide; but it is not, as I regard the case, a controversy on Pasteur vaccination, but on an auxiliary treatment, and in especial on the methods of that treatment. Strictly speaking, cauterization is not a treatment of hydrophobia at all, but a mechanical agent for destroying the poison, subsidiary to the main agent, the specific vaccine.

The principal object then proposed in this paper is to reaffirm in all its rigor and in a pointed manner, the rule that the application of *fuming* nitric acid is the most effectual means of destroying the "virus" of rabies in wounds, and that in all cases in which we fear its coexistence with a bite, it should be got rid of as far and as quickly as may be possible; and further, and above all, to choose from literature and common experience incidents and examples that violate or observe this rule. The principle has already been expressed in plain, emphatic language, in this country by Cabot;[3] in England by Gowers;[4] in Europe by Babes and Lentz.[5] The subject is indeed important. To destroy the "virus" before it has germinated in the wound is the means sometimes of wholly avoiding the disease which it provokes. No more need be said of the technique, which is simple and direct, and entirely adapted to a particularly prompt mode of application. But, it has the high merit of paving the way for the subsequent success of the Pasteur treatment. These are facts determined in more researches than those I have quoted, and in some hitherto unpublished ones by Poor,[6] which I shall quote directly.

It has therefore appeared to me that the endeavor to produce or enlarge this capability of treatment is one of the best services in which a physician can be engaged; and this service, excellent at all times, is especially so at the present day. The reasons of this belief I have already touched upon, but they may be extended as follows: (1) Hospitals, which are apt to prefer the pursuit of some safe and neutral method, furnish almost incredible instances of

[3] Med. News, March 18, 1899.
[4] Manual of Diseases of the Nervous System, ii, 940.
[5] Vorträge u. prakt. Therapie, 1910, 3 s., Heft 10, 762.
[6] Made at the Research Laboratory, Health Department, New York.

bites treated according to convention, the fruitful parent of error; (2) the practitioner in private may be careless from the belief that the case is one for expert rather than ordinary treatment; (3) chemists, apothecaries, and even the newspapers, called by an instinct that gets the better of everything else, exercise not only their own but all other crafts with extraordinary diligence, and accordingly treat bites with those two self-sufficient things—ignorance and irresponsible advice; (4) men who pose as clever in the science of drugs are dissatisfied with caustics, or at least but partially satisfied, hoping to find the ideal form. Much current medicine of a colorless, aimless kind is the work of these purists, whose opinions are wholly influenced by authority. Is this negative method ever likely to command old truths or bring to light new ones?

It is, of course, strict convention to sew up a bite. But this generalized method of treatment has not the happiest results. A striking fraction of deaths from hydrophobia is made up of those whose wounds have been closed with stitches. Viala[7] cites many cases from the Pasteur Institute. How significant is his phrase, "*la plaie est fermée par* 8 (or 6, 3, or 2, as the case may be) *points de suture, et n'a pas été cautérisée?*" It recurs so often in the histories of those who have died from hydrophobia. From such constant uniformity valuable lessons may be drawn. Wounds made by rabid animals should not be sewed up; they should be allowed to bleed. Medically, or scientifically, the point is very serious. The retention of the "virus" destroys the whole balance of safety. For it is a fundamental thing about this disease that a nervous system once infected is not restored by Pasteur treatment. Hence, the use of stitches, instead of caustics, washing and dressing, is a gross and gratuitous blunder; stitches being the means of fructifying the "virus" and very little to the purpose in wounds of this character. It is the destruction of the poison that counts, not the mere healing of a "solution of continuity." The distinction is sound; yet it is overlooked in many hospitals.

In this matter as in all others we have become specialists; the bites of animals cannot be treated as ordinary wounds, even by the most fastidious surgeon, working exquisitely. They do not, it is obvious, require the utmost address; but neither are they to be dealt with according to the accidental tendencies of "schools," "methods," and the like. But worse than any treatment founded on misconception is treatment founded on the notion that any cauterization whatever is of itself useless.

It is a custom that seems a remnant of early medicine to cauterize with carbolic acid. Fermi, among moderns, seems to like it, but Babes[8] has more than once shown its inadequate power.

[7] Annales de l'Institut Pasteur, 1909, p. 512; 1908, p. 558; 1907, p. 486; 1906, p. 511, etc.
[8] Connaissances médicales, May, 1887.

"*Ce virus est peu sensible a l'action de l'acide phénique,*" he wrote long ago, and subsequent experience is confirmatory of this assertion.[9] Sawtschenko,[10] writing in 1911, says: "A solution of phenol, 0.5 per 100, failed to destroy fixed virus, even in twenty days." Fermi,[11] writing a few months later than Sawtschenko, seems aware of the relative weakness of carbolic acid. Of local methods, he adds, the hyperemia of Bier is the most effectual. He applied it, however, only in experiments on mice or rats. Nevertheless, carbolic acid is widely employed as a caustic. I find several instances quoted in the pages of Viala,[12] but only the blank fact that these cases were cauterized with carbolic acid, and that they died of hydrophobia is conceded to us.

The definition, or rule, of treatment, that we are seeking is to be found in the characteristic attributes of nitric acid, namely, its fluidity and caustic power; and the earliest paper that experimentally helps us to determine these attributes is that lucid one by Babes and Talasescu.[13] It is here shown that the Paquelin cautery and the strong mineral acids are of equal power in protecting animals from rabies. This is also the conclusion of Marie,[14] though arrived at in a different way. There are later experiments which show that with cauterization with nitric acid animals may be rendered in many instances secure from rabies. In Poor's experiments, to which I have already referred, 41 per cent. of his animals were saved, while all his controls died. Cabot's figures, derived from experiments with nitric acid, are even higher, but they are less searching, less convincing. Of profound interest is the fact, as all these experiments show, that cauterization is of value as late as twenty-four hours after inoculation. Hence it is that Ravenel[15] writes: "There is strong experimental evidence that the free use of nitric acid will save a certain proportion of cases even after a lapse of twenty-four hours;" and Stimson that: "The best method is to thoroughly touch the wound down to its depth with nitric acid."

Next in importance comes mercuric chloride. Would not washing with a solution of this substance suffice? This is the belief of Hogyes who thinks caustics "illusory." A sense of false modesty shall not prevent us from thinking our own doctrine the better. When we have produced free bleeding, cauterized with nitric acid, and dressed with bichloride, we have done, humanely speaking, the right thing.

Yet so strange are the obliquities of disease, and in particular, of hydrophobia, that those whose minds are much influenced by this fatality will often be tempted to think that there are no fixed

[9] Centralbl. f. Bakteriol., Band lv, 1910, S. 27; Bulletin de l'Institut Pasteur, 1910, p. 677.
[10] Annales de l'Institut Pasteur, 1911, p. 495.
[11] Archivio di Farmacologia sper., 1911, vol. xi, Fasc. iii–iv, p. 152.
[12] Annales de l'Institut Pasteur, 1910, p. 678.
[13] Ibid., 1894, p. 435.
[14] Comptes rendus de la société de biologie, 1907, Tome ii, p. 430.
[15] Hyg. Lab. Bulletin, 1910, p. 52.

principles for its therapeutics to rest on. It is perhaps this spirit which allows the disastrous use of palliatives, iodine, alcohol, and, as one writer recommends,[16] common vinegar.

It is a fact that cauterization of any kind, that is, the application of anything that has the essential quality of caustics, is a valuable means of saving life. According to the writer in Eulenburg's *Real-Encyclopadie*, many are saved in this way in Germany; according to Hogyes cauterization has increased the percentage of cures from 19 to 41 per cent.; according to Proust,[17] "Of 117 people bitten but not cauterized 76 died; while of 249 cauterized only 89 died," all of which shows the high empirical probability of the value of cauterization.

Statistics, it is obvious, do not finally solve problems. Instances gathered in this way make simple calculations of probability not causal connections. It is a method auxiliary to scientific induction so-called, and a causal connection between cauterization and a certain uniformity of result may be inferred by comparing the averages of cases in animals with the average of especially enumerated cases in humans, which are conditioned by the same factor. This process leads us to the possibility of establishing strict uniformity among cases cauterized and cases protected from hydrophobia.

THE MEDICAL ASPECT OF CHRONIC TYPHOID INFECTION (TYPHOID BACILLUS CARRIERS).[1]

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It has proved interesting and instructive in connection with the subject of this paper to review the opinions held by teachers of medicine twenty-five years ago relative to certain infectious diseases. Until about two decades ago studies in the problems of immunity were hardly begun, while the science of bacteriology was in its infancy. In fact, because of the rapid development of the science rather than the art of medicine, it may be safely asserted that more progress has been made in our understanding of the early and late effects of infectious diseases during this period than had been possible during the centuries preceding. This progress has been made possible through the labors of the early investigators, and it is more fortunate for the coming generations than we can

[16] Töpfer, Klin. Jahrb., 1907.

[17] Annales de l'Institut Pasteur, 1894, 435.

[1] Read before the Mississippi Valley Medical Association at Nashville, Tennessee, October 17, 1911.

conceive that there existed such creative minds as those of Koch, Pasteur, Virchow, Ehrlich, and Wright.

We now understand, tentatively at least, many facts not available even a decade ago regarding the late effects of certain infections. The early effects were more readily recognized clinically, while less importance was attached to the remote sequelæ except in so far as certain complications retarded individual recoveries. Little was thought then of the late effects of certain infections upon the mass-hygiene or sanitary condition of communities. For example, one decade ago but little importance was attached to the bacteriological condition of the child's throat subsequent to diphtheria, and the importance of the subject was not appreciated. It was hardly considered possible that many such children became chronic carriers of the infection for weeks or months subsequent to their attack. We now realize that a large percentage of the disease incidence arises in others from exposure to the mouth and throat secretions of such individuals. Nor in tuberculosis was it generally recognized that many individuals with chronic non-active types of the disease seryed as disseminators of the infection for periods of years, while themselves able to enjoy the blessings of apparent health.

Likewise, in typhoid fever, an ever present disease in our American municipalities, and in Asiatic cholera, the remote effects of these infections have only recently been appreciated. It is now generally recognized that about 4 per cent. of all typhoid cases become chronic carriers and disseminators of the infection for periods of months or years after their apparent recovery. It is entirely possible for the human body to harbor disease microörganisms of many other types for long periods of time without interfering with the apparent health of the individual. Sternberg discovered the micrococcus of pneumonia in healthy sputum, and every physician is familiar with the latent forms of syphilis and tuberculosis, which for long periods remain quiescent, and do not lead to destructive tissue changes. The various factors of immunity which protect the individual in such instances are not understood, but are generally covered by the explanation, so frequently given, but which serves merely to cloak our ignorance, the resistance of the individual and the varying virulence of different strains of bacteria.

In considering typhoid fever we have to deal with the following two modes of dissemination:

1. The spread of the disease by means of water, milk, and food products contaminated directly or indirectly by some active typhoid infection, but, as a rule, *remote* from the source of infection.

2. By contamination of water, milk, or food products *directly;* that is to say, by typhoid bacillus carriers who are in some way connected with the handling of food supplies.

We recognize two types of chronic typhoid carriers—"contact,

idiopathic or primary carriers," who have never had the disease to their knowledge, and "secondary carriers," who have at some time previously had the disease.

PRIMARY, IDIOPATHIC, OR CONTACT CARRIERS. Typhoid carriers who become such through association with the disease, and such instances are not uncommon among nurses and orderlies, but who themselves have not had typhoid to their knowledge, are called "contact carriers." Since every typhoid patient requires the constant care of at least two or three individuals, it may readily be surmised that a large percentage of such attendants become "contact carriers." In many instances such individuals continue to excrete typhoid bacilli in the stools and urine for periods of years. Their own tolerance to the infection is probably due either to natural immunity or to a partial immunity from an earlier unrecognized mild typhoid infection.

Houston,[2] in 1899, reported the first case of persistent typhoid bacilluria in a contact carrier, who for three years had shown symptoms of chronic cystitis, while Drigalski and Conradi,[3] in 1902, found typhoid bacilli in the stools of 4 persons who had had no previous typhoid fever symptoms, but who had been in contact with typhoid fever. Park[4] has estimated "that probably one in every 500 adults who has never knowingly had typhoid fever is a typhoid-bacillus carrier." At first glance such figures are appalling, but with our recent knowledge of so-called "short duration typhoid" and of other factors, such as individual resistance and the varying virulence of different strains of bacteria, we can appreciate how many individuals at some time during their lives suffer an active typhoid without themselves being aware of it. In fact, it is entirely possible that many so-called summer diarrheas are in reality mild typhoid infections, which are not so diagnosticated. Such individuals receive a partial immunity, partial in the sense that, while themselves protected against the damaging influence of the infection and experiencing little subsequent disturbance, they are able to harbor, excrete, and perpetuate the bacilli in large numbers. Bacilli from such individuals when engrafted upon non-immune soil are able to produce all the clinical evidences of a severe attack.

SECONDARY CARRIERS. As above mentioned about 4 per cent. of all individuals who have, to their knowledge, had typhoid become chronic carriers of the disease for periods of months or years. They have secured from the previous attack only a partial immunity in the sense that while certain protective immune bodies, such as the opsonins and stimulins, are augmented by the attack, others, such as the bactericidins and bacteriolysins, for some

[2] Brit. Med. Jour., January 14, 1899 ibid, October 9, 1909.
[3] Quoted by Park, Jour. Amer. Med. Assoc., September 19, 1908.
[4] Jour. Amer. Med. Assoc., September 19, 1908, p. 981.

unknown reason, are not augmented to the point of destruction of the bacilli. In other words, the factors which protect them individually from further damage do not lead to complete destruction of the infection, that is, their immunity is phagocytic, not bactericidal or bacteriolytic. Experience has shown that many typhoid bacillus carriers experience comparatively little discomfort. The attention of the physician is secured either because of their probable connection with endemic outbreaks or from individual symptoms, vague in character which lead to an investigation.

Fütterer[5] in 1888 first called attention to the presence of typhoid bacilli in the gall-bladder, while Gilbert and Giroche, in 1890, are generally credited with the first description of typhoid bacillus cholecystitis. In 1893 Chiari[6] was able to collect 13 instances of typhoid bacillus cholecystitis and, in 1898, Cushing[7] tabulated 6 cases. The articles by these investigators and the paper by Ehret and Stolz[8] in 1900, in which 32 cases were described, had much to do with the recognition of the importance of the subject. Much discussion has arisen concerning the way in which typhoid bacilli reach the gall-bladder. The recent work of Chiari,[9] Hirch,[10] and Forster[11] and of Foster and Kazsen, would seem to convince most of us that the infection reaches the gall-bladder, in most instances, from the liver secretions or by way of the blood stream. Such mode of transmission undoubtedly occurs in contact carriers who have had a typhoid bacteriemia without the clinical evidences of enteritis, for Busse[12] has shown that the presence of typhoid bacilli in the blood stream do not necessarily give rise to enteritis or, for that matter, to agglutinating powers toward the bacilli. On the other hand, there is considerable evidence to show that in acute typhoid enteritis the bacilli may reach the gall-bladder from the duodenum. That typhoid ulceration may occur in the duodenum and stomach cannot be doubted. Such an instance of perforation of a typhoid ulcer in the duodenum occurred in the practice of my colleague, Dr. L. C. Grosh, in 1907.

After an attack of typhoid the infection may remain latent in the gall-bladder or in the alimentary or urinary tract for periods of years. In fact, with an earlier typhoid history, it is improbable that an acute exacerbation of latent cholecystitis is anything else but the result of the earlier infection. Levy and Kayser[13] found typhoid bacilli at autopsy in the gall-bladder of a patient who had no symptoms of cholecystitis, but who had had typhoid fever three

[5] Münch. med. Woch., 1888, p. 315.
[6] Präger med. Woch., 1893, p. 1.
[7] Johns Hopkins Hosp. Bull., 1898, ix, 91.
[8] Mitteilungen a. d. Grenzgebieten d. Med. u Chir., 1900, p. 389.
[9] Verhand. d. Deutsch. patholog. Gesellschaft, 1907, xi, 143.
[10] Ibid., xi, 150.
[11] Verhand. d. Deutsch. patholog. Gesellschaft, 1907, xi, 160.
[12] Münch. med. Woch., 1908, lv, No. 21.
[13] Ibid., 1907, liii, No. 50.

years previously. Grimme[14] has reported the case of an insane patient who was isolated because of the presence of typhoid bacilli in the stools, and from whom numerous gallstones were removed at operation. After an interval following the operation, typhoid bacilli were no longer found in the stools. Hammond[15] found typhoid bacilli in the gall-bladder one year after an attack of typhoid. Hamilton,[16] who investigated 24 individuals with history of gall-bladder disease, found that 2 were typhoid bacillus carriers and 5 paratyphoid carriers. Fromme[17] has reported 4 cholecystectomies in 4 women typhoid carriers in all of whom elimination of bacilli ceased following the operation. Billings[18] has described the conditions present in a contact carrier who gave a positive Widal reaction and from whose stools a pure culture of typhoid bacilli was obtained. Upon operation for cholecystitis, calculi and typhoid bacilli were found.

Holmes'[19] recent article on the subject also shows that cholecystitis due to typhoid bacillus can occur, as in one of Cushing's[20] patients, without the history of an earlier attack of typhoid. Such patients must be considered contact carriers. In Holmes' patient the bacilli disappeared from the bile and stools in eight weeks following cholecystostomy. Holmes has very properly brought up the point that the mere presence of typhoid bacilli in the gall-bladder is not of itself sufficient to provoke the clinical symptoms of cholecystitis. It is only when the cystic or common duct is obstructed, during perhaps an otherwise latent period of the gall-bladder infection, that symptoms manifest themselves which we recognize as the clinical evidences of cholecystitis.

In most chronic carriers the bacilli are eliminated with the feces; in a smaller number the urine contains the bacilli. Forster believes the gall-bladder to be the site of continued reproduction of the bacilli which are intermittently ejected into the intestine. The Ledinghams uphold this view, since investigations have shown that the bacilli may disappear during the convalescent period of the disease only to reappear several months later, following perhaps, the administration of some cholagogue cathartic as suggested by Reitter.[21]

The History of Typhoid Bacillus Carriers. Experience has shown that, in most cases, the discovery of typhoid carriers has arisen from hitherto unexplainable endemic outbreaks among the inmates of asylums, or among families who have happened to

[14] Münch. med. Woch., 1908, lv, No. 1.
[15] Jour. Amer. Med. Assoc., January 2, 1909.
[16] Ibid., February 26, 1910.
[17] Deutsch. Zeitschr. f. Chirurg., 1910, cvii, No. 6.
[18] Trans. Amer. Med. Assoc., June 27, 1911; abst. Medical Record, New York, 1911, lxxx, No. 1.
[19] New York Med. Jour., August 12, 1911.
[20] Loc. cit.
[21] Wien. med. Woch., 1908, p. 519.

employ the same cook, or obtained milk from a dairy previously under suspicion. A large number of typhoid carriers, in the sense that they have been the cause of such endemics, have been women who were, in some way, connected with the handling of food products. On the other hand, many carriers exist who, rather from good luck than good management, do not happen to infect others. Any individual who has been in contact with the disease, or who has himself suffered an earlier infection, may be a carrier in the sense that pure cultures of the bacilli may be obtainable from the stools or urine. Probably not more than one-half of such carriers actually infect others, although no definite data is available on this point, since the matter of cleanliness in personal habits varies, as a rule, with the social status of the invividual.

Until the recent communication of Young, mentioned below, the largest number of recorded cases originating from one source was reported by Lumsden and Woodward,[22] who found a typhoid carrier in a dairy responsible for 54 cases of the disease among the dairy customers. This carrier had had her attack eight years before. The time element since the original typhoid attack has been found to vary greatly. Gregg[23] has reported the case of a boarding-house mistress, who served as the source of infection in 7 cases of typhoid, fifty-two years after her own recovery. No bacilli were found in the blood or urine, but pure cultures were obtained from the feces. Jundell[24] has described the conditions present in a family, the grandmother of whom was known to be a typhoid carrier for fifty-four years, and during this interval 22 members of the family were attacked. This carrier was eighty-three years old, and her feces were found to contain typhoid bacilli.

Scheller[25] has described an endemic in Prussia in which during a period of fourteen years, 32 cases of typhoid were traceable to a woman employed in a dairy whose attack had occurred seventeen years previously. Typhoid bacilli were found in her stools in pure culture. Out of 40 people who drank the milk of this dairy, 18 were found to be carriers of the infection, and yet only 5 of the 18 had ever had typhoid. In other words, 13 were contact carriers. Soper's[26] painstaking investigation of so-called "Typhoid Mary," a contact carrier, showed that in ten years, 26 cases of typhoid could be traced to her as cook in households where the disease appeared. Typhoid bacilli were found in the stools, but none in the urine.

Young,[27] Commissioner of Health, of Chicago, in a personal

[22] Jour. Amer. Med. Assoc., March 6, 1909, p. 749

[23] Boston Med. and Surg. Jour., July 16, 1908.

[24] Hygiea Festband, 1908; abst. edit. Jour. Amer. Med. Assoc., January 30, 1909, p. 388.

[25] Centralbl. f. Bakteriol, 1908, No. 5, p. 385; abst. edit. Jour. Amer. Med. Assoc., June 13, 1908, p. 1986.

[26] Jour. Amer. Med. Assoc., June 15, 1907.

[27] Personal Communication, July, 1911.

communication, has recently described the conditions which resulted from the employment of a young woman in one of the suburban dairies. In June and July, 1911, three years after her apparent recovery from typhoid, 56 cases of typhoid were found in families receiving their milk supply from this dairy. To July 21, 1911, 4 deaths had occurred among this number. Negative bacteriological results were obtained in the study of the bowel and bladder discharges of all individuals connected with the dairy, except in the case of the young woman above mentioned. Part of her duty was to wash the milk cans. Nieter and Liefmann[28] found 7 carriers among 250 inmates of an insane pavillion where typhoid was endemic. At the autopsy of 1 patient who succumbed to chronic dysentery, typhoid bacilli were found in the intestines and in pure culture in the gall-bladder. The serum of this patient would agglutinate the typhoid bacillus in dilution 1 to 50, and the Flexner dysentery bacillus in dilution 1 to 100. After isolation of the carriers, the endemic ceased. Busse[29] was able to locate typhoid bacilli from the blood of 4 carriers who died of intercurrent diseases. Hutchinson[30] has reported the career of a lodging-house keeper whose attack occurred fifteen years earlier and who was responsible for 7 cases of typhoid. Similar investigations have been made by the Ledinghams,[31] by Connell,[32] Huggenberg,[33] Southward,[34] Dehler,[35] Nieter,[36] Branthwaite,[37] and by Madsen.[38]

DIAGNOSIS. It will be obviously impossible to keep any large number of typhoid convalescents under bacteriological surveillance. As mentioned in an earlier article[39] popular education may be of service in dealing with the problem. This education can best be carried on by physicians who should bear in mind the possible connection between an earlier attack of typhoid, or the possibility of infection by contact, and symptoms, sometimes slight, referable to the gall tract, to a slight urinary cystitis, or bacteriuria, to chronic colitis, to mild dysentery, or to attacks of so-called intestinal indigestion. The source of endemic outbreaks should be investigated whenever possible, and the possibility of direct infection of food products by a typhoid bacillus carrier should be borne in mind. Surgeons should have cultures taken in all operations involving the gall-bladder, and physicians at the time of their examinations

[28] Münch. med. Woch., 1906, liii, No. 33.
[29] Loc. cit.
[30] Brit. Med. Jour., March 26, 1910.
[31] Ibid., January 4, 1908; ibid., October 17, 1908.
[32] AMER. JOUR. MED. SCI., 1909, cxxxvii, 637.
[33] Correspbl. f. Schweiz. Aerzte, 1908, xix, 635.
[34] Archives of Pediatrics, March, 1907.
[35] Münch. med. Woch., 1907, liv, No. 16; ibid., 1907, liv, No. 43.
[36] Ibid., 1907, liv, No. 33.
[37] British Government Report, abst. Jour. Amer. Med. Assoc., April 4, 1908.
[38] Hospitalstidende, Copenhagen, 1910, liii, No. 14; abst. Jour. Amer. Med. Assoc., June 25, 1910.
[39] Jour. Amer. Med. Assoc., 1910, lv, 1708.

should search for post-typhoidal sequelæ in those who have had the disease, and for contact carriers through association, in those who deny previous infection. Such sequelæ are especially apt to be found among patients presenting evidences of gall-bladder disease, the symptoms of which are frequently referred to the stomach. In fact, all patients showing evidences of gall-gladder disease, whether months or years after a history of typhoid, should be considered as possible chronic typhoid carriers until proved otherwise.

Cultures should be taken in all post-typhoid cases whose urine contains bacteria. Likewise cultures should be taken from the stools in all cases showing symptoms of chronic dysentery or colitis. The colon bacillus will practically always be obtained from the stools, and may be confusing, but the typhoid bacillus, if present, can be isolated from the plates with comparatively little difficulty. The diagnosis will depend upon the isolation of a bacillus which gives culturally the main characteristics of the bacterium of Eberth. That is to say, a short motile rod which grows rapidly in bouillon or on agar slant, which does not liquify gelatin nor produce indol or coagulate milk, nor produce gas or acid in glucose litmus gelatin, and produces an invisible growth on potato.

It is the personal conviction of the writer that these main characteristics are not given by all strains of typhoid bacilli, for in 1 of the 3 cases studied during the past two years the cultural findings were confusing. In one (C. R. D.) who had never had typhoid and who was regarded as a contact carrier, the pure cultures obtained from the urine throughout a long period of treatment, would at times correspond to the main cultural characteristics mentioned, while again slight amounts of gas and acid would be produced in the media. In this connection it may be worth mentioning that no symptoms of cystitis or pyelitis were present in this patient. He was referred for examination because of the constant presence of enormous numbers of bacteria in the urine. During many months of treatment no pus cells were found, a condition seldom, if ever seen, when the colon bacillus is the causative organism.

In other words, the colon bacillus when present in the bladder produces an inflammatory reaction which is recognized as a true cystitis, while the typhoid bacillus when present in the urinary tract, in typhoid carriers is eliminated in suspension in the urine, a condition recognized as bacteriuria. Still further proof, in support of the view that this patient was a typhoid carrier, is afforded by the fact that he failed to react to the treatment ordinarily successful in dealing with colon bacillus infections of the urinary tract; that is, the use of hexamethylenamine and sodium benzoate internally and the use of a colon vaccine. It may be mentioned also that his blood serum would agglutinate, in dilution 1 to 50, a stock typhoid culture. He was not benefited by inocu-

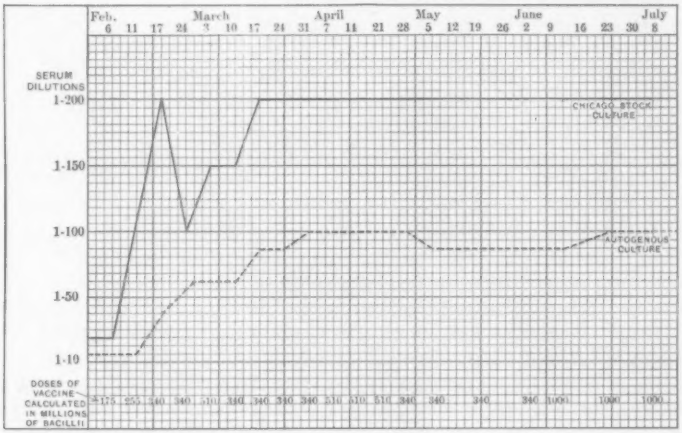
lations of an autogenous vaccine, and gave up treatment before inoculations of a stock typhoid vaccine were begun.

The agglutinating power of the blood serum as well as the phagocytic power is found to be increased in typhoid bacillus carriers, as a rule, while the bactericidal and bacteriolytic powers are below normal. This is in accord with the previously expressed opinion that such individuals are but partially immune, since it has been shown[40] [41] that normally during convalescence from the disease, the bactericidal and bacteriolytic properties of the serum are increased to the point of destruction of the bacilli. These facts are of value in the bacteriologic study of a suspected individual and may be of service from a diagnostic standpoint. Alice Hamilton[42] has also shown that carriers have a persistently high opsonic index and relatively high agglutinative power; that is, their immunity is phagocytic, not bacteriolytic, or bactericidal.

Report of a Typhoid Carrier and Vaccine Treatment. R. F. (record 565), aged twenty-eight years, from Deerfield, Michigan, was referred by Dr. R. M. Eccles, January 17, 1911, as a possible typhoid carrier, although no cases of the disease were traceable to him. In 1904 this patient had typhoid fever for six weeks with apparent recovery for one year, after which time he began to complain of pain in the region of the umbilicus which continued until 1909. The subsequent history was supplied by Dr. Geo. Sellards, of Deerfield. In 1909, diarrhea began; the bowel discharges numbered five or six daily with some mucus, and in December, 1909, three severe hemorrhages from the bowel occurred, with temperature as high as 104°. The last hemorrhage measured over one quart. The diarrhea continued for one year thereafter. The discharges were occasionally streaked with blood and mucus but gradually subsided under medication. The weight before the attack of typhoid seven years ago was 151. At the time of examination it was 134. He complained of frequent headaches. The examination revealed nothing of importance on the part of the heart, lungs, nervous system, and abdomen, except some tenderness on pressure was present below the umbilicus. One loose stool occurred every morning, with poor control of the sphincter ani. Examination of the fresh stool for Amebæ dysenteriæ, as well as stains of the mucus present for tubercle bacilli were negative. The von Pirquet skin reaction was negative. The leukocytes numbered 7200, and the urine was negative to albumin, pus, and sugar. There had never been any urinary symptoms. The Adler occult blood test was positive in the stools. Cultures taken from the stools showed the presence of the colon bacillus and a short, plump, actively motile rod, which in pure culture, did not produce

[40] Proc. Royal Soc., 1903, and Jour. Royal Army Med. Corps, 1907 and 1908.
[41] Jour. Amer. Med. Assoc., 1909, liii, 1253.
[42] Loc. cit.

acid in litmus gelatin, or gas in stab culture on glucose agar, nor was indol produced after ten days growth in bouillon. Milk was not coagulated. The agar slant showed a growth which resembled that of the typhoid bacillus, while the growth was invisible on potato. Gelatin was not liquefied in six weeks. Cultures from the urine showed a few isolated colonies of Staphylococcus albus (probably contamination). Subsequent cultural studies of the fresh stool gave the same findings. His blood serum would agglutinate a stock typhoid culture in dilution 1 to 20, while the autogenous strain would agglutinate in dilution 1 to 10.



Mr. R. F., aged twenty-eight years. Chart shows the increase of the agglutinating power of his serum for the bacillus of Eberth under injections of autogenous typhoid vaccine.

A vaccine was prepared from the typhoid organisms present in the stools. He received his first inoculation on February 6, 1911. Between that date and May 5, 1911, fourteen inoculations were given at weekly intervals, the doses varying between 110,000,000 and 510,000,000. Negative cultures were obtained in May, 1911. Since that time he has received five additional inoculations, three of which have contained 1,000,000,000. The agglutinating power of the patient's serum was increased ten fold during the course of treatment. Coincident with the vaccine treatment and the increase in the agglutinating powers of his serum (see Chart) his general condition improved. The tendency to loose stools with poor sphincter control disappeared and the bowel discharges became normal in appearance and gave negative cultures. He was discharged July 8, 1911.

THE MEDICINAL TREATMENT AND CONTROL OF TYPHOID CARRIERS. The problem is a perplexing one, since the installation of adequate filtration plants and careful dairy supervision will not, for years to come, rid most American municipalities of typhoid. In most localities the disease has been endemic for years with a consequent large number of unrecognized carriers. Such individuals if employed as cooks, waiters, and waitresses, dairymen and dairymaids, workers in confectionery, hucksters, workers in artificial ice plants, or in any capacity which requires the handling of food products may continue to serve throughout long periods of time as a menace to the public health.

Since the treatment of such individuals has not been uniformly successful, the question immediately arises as to what steps local and state boards of health should employ to protect the public. For example, in the recent Chicago outbreak, 56 cases of typhoid with 4 deaths have resulted from the uncleanly personal habits of a young woman employed to wash the milk cans in the dairy. What other attitude could or should be taken by the Municipal Department of Health except to employ its police powers toward the isolation of such an individual for treatment. Although innocent of harm she has been directly responsible for 4 deaths to date, to say nothing of the invalidism and expense incurred by over 50 others. There is much to be said regarding personal liberty, but since the law gives protection to property, personal and real, it is only fair to assume that such protection should be given to the health of individuals who are well; an asset far greater than the asset of property. Progress in every walk of life depends upon those who are in good health, but strange to say, the wheels of progress turn slowly, and stranger still, our law-making bodies can be induced to attach relatively little importance to prophylactic medicine. However, the danger to the public health will be greatly minimized, if when typhoid bacillus carriers are discovered control could be exercised over them by the local boards of health, with the idea of securing proper treatment, and restricting them to employment which do not have to do with the handling of food supplies.

The treatment of the chronic typhoid carrier by so-called intestinal and urinary antiseptics, such as sodium phenolsulphonate, sodium salicylate, salol or hexamethylenamine, and sodium benzoate, has been of little avail. During convalescence from the disease when the natural immune bodies, such as the bacteriolysins and bactericidins, are augmented, such antiseptics are undoubtedly of value and should be employed in every case as an useful adjunct. Graham, Overlander, and Dailey[43] found that 23 per cent. of 65 typhoid patients showed typhoid bacilli in stools and urine for ten

[43] Boston Med. and Surg. Jour., January 14, 1909.

days before their discharge. An important lesson should be learned from these facts, since it is entirely probable that if the convalescence of typhoid cases was studied and treated with as much care as the active stages of the disease, the number of chronic carriers would be greatly lessened. During convalescence from the acute attack, when conditions are most favorable because of the natural augmentation of immune bodies, help may be secured by medicinal means; while in chronic carriers the bactericidal and bacteriolytic properties of the blood serum are decreased and such medicinal assistance as may be offered, is of little avail.

VACCINE TREATMENT. The number of typhoid carriers successfully treated by vaccines has not been large. Irwin and Houston[44] were able to cause disappearance of the infection in a patient who for seven years had been a carrier. She received during two months, five injections varying from 50,000,000 to 500,000,000. Meader[45] has also successfully treated a patient and found that the best therapeutic dose varied between 75,000,000 and 400,000,000. He has shown that the disappearance of the infection coincided with the increase in the bactericidal power of the blood serum of the patient. In my report[46] of a typhoid carrier treated during 1910, it was shown that the agglutinative, bacteriolytic and bactericidal properties of the blood serum were augmented during the course of six injections which varied from 100,000,000 to 400,000,000. The increase in the immune bodies of the serum was followed by the disappearance of the infection. Brem and Watson[47] have recently reported the case of a child, aged four and one-half years, whose urine was found to contain the bacilli a few months after convalescence. She subsequently received nine injections of autogenous vaccine in about two months, when the urine was found to contain no bacilli. The agglutinative powers of the patient's serum increased markedly to 1 in 1000 during the course of inoculations.

In fact, it may be said, that the treatment of carriers by inoculations of vaccine offers more hope for cure than any other known method. It probably is better to use a vaccine prepared from the patient's own organisms, although no definite proof exists that such is the case. The work of Leishman and his associates seems to prove that as efficient an immunity is conferred by a stock vaccine as by an autogenous vaccine, and that a single nonvirulent strain is capable of producing as efficient an immunizing response as vaccine prepared from single or multiple virulent strains. The method of dosage found to be most satisfactory has been to increase gradually from 100,000,000 to 1,000,000,000 depending upon the

[44] Lancet, London, January 30, 1909. [45] Johns Hopkins Hosp. Bull., September, 1910.
[46] Loc. cit.
[47] Trans. Amer. Med. Assoc., June 27, 1911; abst. Medical Record, New York, 1911, lxxx, No. 1.

evidences of immunizing response as obtained by study of the agglutinating, bactericidal, and bacteriolytic power of the blood serum.

The vaccine should be prepared as follows: Pure cultures of the organisms are grown on agar for twenty-four hours, when the growth is washed off with sterile salt solution. The resulting suspension is thoroughly shaken to break up the clumps and a small quantity drawn into an ordinary red blood pipette, diluting 1 to 200, with salt solution for purpose of standardization. A small drop of this dilution is then placed upon the *blood-platelet* counter of Zeiss. The *blood-platelet* counter differs from the ordinary red blood cell counter in that with the former, the depth of the chamber is only $\frac{1}{50}$ mm. Twenty-five small fields are counted under the microscope, using dim direct illumination or indirect illumination by the dark-field condensor, and the sum divided by twenty-five to obtain the unit value. The unit value is then multiplied by the dilution, by the depth of the chamber, by 400, the number of small squares in a millimeter, and, lastly, by 1000 to convert to cubic centimeters. For example, if fifty bacterial cells are counted in twenty-five small squares, the unit value per square would be two, multiplied by 200, by 50, by 400, and by 1000, or 8,000,000,000, the number of bacteria per c.c. of suspension. The suspension is then heated to 54° to 55° C. for one hour in a sealed test-tube, in a water bath and then diluted with sterile salt solution, containing 0.35 per cent. phenol, so that each cubic centimeter of the finished vaccine contains approximately 1,000,000,000. Care must be exercised not to overheat the vaccine, since overheating impairs its immunizing properties. The thermal death point of most strains of typhoid bacilli varies between 53° and 56° C. Cultures should be taken from the vaccine to insure sterility before using.

Conclusions. 1. Not only should individuals who are typhoid carriers receive appropriate vaccine treatment, but all individuals including physicians and nurses, whose duties require the attendance and care of typhoid cases should receive immunizing treatment. By this procedure many individuals otherwise destined to become chronic carriers might secure protection which would prevent in themselves the possibility of cholecystitis or other late manifestations, as well as avoid the possibility of becoming a source of danger to others.

2. Such treatment has been found efficient and productive of little discomfort. Nurses, orderlies, and physicians should be regularly immunized at intervals of two or three years, as should all others exposed to endemic or epidemic outbreaks.

3. When typhoid bacilli have produced clinical or bacteriological evidences of cholecystitis, it is doubtful whether vaccine treatment will of itself be of avail, surgical drainage of the viscus then being indicated. On the other hand, appropriate vaccine treatment in

individuals showing evidences of intestinal or urinary tract perpetuation of the bacilli, without cholecystitis, during convalescence from the disease or subsequently, should be given the opportunity of vaccine treatment. Not all will be cured but the bacilli will disappear in some instances.

4. Chronic carriers who have been unsuccessfully treated by vaccines should be under the control of the local board of health and furnished employment if necessary which would not require the handling of food products.

EPIDEMIC POLIOMYELITIS: A CLINICAL STUDY OF THE ACUTE STAGE.[1]

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It is an interesting fact, that although our knowledge of acute poliomyelitis dates back over seventy years, when the German surgeon, Jakob Heine[2] first described it under the name of "the spinal paralysis of children;" the true nature of the disease, its pathology, clinical manifestations, prognosis, infectiousness, contagiousness, have only lately been fully ascertained, and have, only in the last decade, become the subject of general knowledge. Furthermore, it is of interest to note that at the same time that Heine first made his observations in Germany, an American practitioner, Dr. Colmer, observed a small epidemic in Louisiana, and recorded it in the Amer. Jour. Med. Sci. (1843, v, 248). Little or nothing was added to the knowledge of the subject for the next thirty years. No epidemics were reported until 1881, when 18 cases occurred in Sweden. Subsequently, epidemics were reported from Norway, Sweden, Germany, the United States, and Canada, in the eighties and nineties. The most extended epidemics occurred in the above-named countries in the last five or six years.

A noteworthy contribution was made by Strümpell[3] in 1885, in his paper on Poliencephalitis Acuta in Children. Pathologically and clinically this disease was noted as identical with the well-known forms of spinal poliomyelitis. For the first comprehensive clinical studies we are indebted to the Danish physicians Medin[4] and Wickman.[5] Aside from his contributions to the pathology, Wickman was able to show conclusively, that the disease is contagious.

[1] Read before the Metropolitan Medical Society, Academy of Medicine, October 24, 1911.

[2] Die spinale Kinderlaehmung, 1840 and 1860: a study of 150 cases.

[3] Jahrb. für Kinderheilk., 1885, Band xxii, N. F.

[4] X. Internationaler Congress, Berlin, 1890.

[5] Beiträge zur Kenntniss der Heine-Medinschen Krankheit, Berlin, 1907.

Pathologically speaking, the disease has been characterized as a meningo-encephalo-polio-myelitis. Of late, it has been shown, however, that the lesions are not confined to the gray substance of the brain and cord, but may be found in the contiguous white substance, as well as in parts which have no direct communication with the gray matter.

Recently the study of poliomyelitis has entered into a new phase through its successful inoculation into monkeys.

In taking up this subject of poliomyelitis I wish to dwell mainly on its diagnostic features. If this be an infectious and contagious disease, as now seems certain, our duty as practitioners clearly lies not only with the patient, but also with the family and the community. Aside from the fact that the recognition of its inception is of great importance on account of the dubious prognosis as to life and limb, it is clearly our duty to diagnosticate it as early as possible, in order to prevent its transmission to others.

After a careful study of the manifold clinical manifestations leads up to a diagnosis of the case, the question of prognosis is the all important consideration. Having this in view, I would therefore, propose the following classification, which, empirical though it may seem, is comprehensive, and tends toward a practical purpose. Poliomyelitis can be divided into: (1) The abortive cases, mild or severe in their onset or course, which end in complete restitution in a short time; (2) cases with paralyses, these may end in complete restitution or in partial or complete atrophy; (3) the rapidly fatal cases (formes frustes). This classification has some value, because it covers the whole ground and lays equal stress on the mild, transient forms, as well as on the paralytic and the fatal cases, putting the former on a par with the well-known paralytic forms.

By this classification I mean to lay stress on the fact that a large number of abortive cases pass unrecognized at the present time, while, judging from our experience of other infectious diseases, these same abortive cases spread the epidemic. Observations made during epidemics of poliomyelitis seem to show that the group of cases occurring not infrequently in infants and young children, hitherto styled meningitis serosa, as well as certain unclassified spastic or tetanic affections of childhood may well belong to the abortive type of poliomyelitis. This also holds good for certain unexplained fatal cases in children and adults. How large their number is, however, cannot at present, be estimated.

What means are at our disposal at the present time to recognize with any degree of certainty a case of acute poliomyelitis? What are the characteristic symptoms of the disease; what additional diagnostic aid does the laboratory offer toward making a positive diagnosis?

In studying the literature of the extended epidemics of Sweden, Norway, and Germany, it becomes at once apparent that the recognition of cases, even though they may be atypical in their clinical manifestations, seems comparatively easy. The existence of a widespread epidemic has been officially noted, the authorities are on the alert for every case, the practitioner is instructed to watch for and report all suspicious cases. An organized medical campaign is thus established in the country districts, the hamlets, villages, and small towns, and the progress of the epidemic can be followed from house to house.

In a city like New York, for example, conditions are totally different. It is true that we are forwarned that the epidemic generally appears in the summer months, and, furthermore, we know that the disease has become endemic with us. Thus, sporadic cases make their appearance at any time, and must be recognized without the aid of epidemiological data. If the cases are brought in from the country, it is sometimes possible to determine the existence of an epidemic in that outlying district. If the case is not an extraneous one, a clue as to its origin and transmission, in the city proper, can usually not be obtained, thus the etiological factor does not become apparent, and the possibility that one is dealing with a case of poliomyelitis is not even thought of or considered. Nothing would be of greater aid to the practitioners of large cities than the daily publication in the public press, by the Board of Health, State or National Health Boards of the daily number of cases of infectious and contagious diseases. In this way the family doctor would be apprised of the existence, the increase and decrease of the various epidemics, and exact information on these points would materially aid him in the diagnosis of suspicious cases.

Since Medin's time, clinicians are wont to look for the predominance of certain irritative and paralytic symptoms of the nervous system, to aid them in recognizing the disease in its various forms. Aside from the abortive cases, which may present themselves under the most varied guise, the principal types of poliomyelitis are: The spinal, meningeal, cerebral, encephalitic, bulbar, and pontine, Landry's ascending and descending paralytic, the polyneuritic, and the ataxic types. It goes without saying, that these various types may overlap or combine. There is no room for doubt, that the systematic subdivision into the above-named groups, indicating as they do, the seat of the lesion, lend material aid in the diagnosis of poliomyelitis in its paralytic stage. In the acute stage, however, they only serve the purpose of indicating the existence of some infectious process in the cerebrospinal system, the nature of which must be especially determined. In other words, our method of diagnosis, at the present time, is a purely empirical one, and will remain so, until the diagnosis can be made from the secretions or excretions, from the blood or spinal fluid, bacterio-

logically, serologically, or by other exact biological methods. Our method of diagnosis, then, being purely empirical it is just as important to note certain other symptoms, which, though they may vary in different epidemics, must be carefully considered, as they are present at the very inception of the attack and may cover up or disguise the true nature of the underlying affection. A poliomyelitis may begin with or run its course under the form of an angina, tonsillitis, influenza, a gastro-enteritis, a rheumatic fever. In some epidemics most of the cases were accompanied by the various respiratory and intestinal symptoms of acute influenza. Indeed, a Swedish author[6] has attempted to show that poliomyelitis is only a form of influenza infection, although he has failed to demonstrate the constant presence of the bacillus of Pfeiffer in his cases. Angina and tonsillitis are often seen in the prodromal stage, or are the concomitant symptoms of the early acute stage. Again, the gastro-intestinal tract seems to be the principal seat of the infection in some epidemics, and the disease runs its whole course under the mask of a diarrhea accompanied with fever. The description of such a case is given below. When these cases occur in an abortive form, it is doubtful whether they can be recognized at all, especially in infants and young children, unless a carefull search is made for symptoms of irritation of the brain and cord.

The Abortive Cases, Their Diagnosis and Significance. Our knowledge of the abortive cases of poliomyelitis only date back to the observations of Wickman[7] made in the great Swedish epidemics of 1905 and 1906. He holds that they are frequent, probably exceeding in number the paralytic cases. As they have hitherto only been recognized when they occur in connection with typical cases, it follows that the number of abortive cases is much greater than is ordinarily supposed. Their epidemiological importance cannot be overestimated. Wickman cites a large series of family infections, in which abortive cases occurred in connection with paralytic cases of varying intensity, together with fatal cases. Some of the children after a short feverish attack were well in a few days or a week, and returned to school. Accordingly, the abortive cases are the principal carriers of the infection.

In my service at the German Hospital, in September, 1910, an abortive case of poliomyelitis of the gastro-enteritic type came under my notice, which was not recognized during its course, but was subsequently proved to belong to this group, through the kind interest of Dr. Simon Flexner. The case was published in the *Medical Record*, of July, 1911. As it is very instructive, I will mention it here, somewhat in detail:

[6] Th. Brorström, Akute Kinderlachmung und Influenza, Leipzig, 1910.
[7] Die akute Poliomyelitis, Berlin, 1911.

CASE I.—J. W., aged six years, entered the Hospital September 4, 1910, discharged cured, September 21, 1910. Diagnosis, acute dysentery, meningism. His father was living. His mother died of consumption. He had measles in February, 1910. For the several weeks before admission he had been restless at night. On September 3, at noon he was seized with a chill, then complained of headache, which gradually became very severe. He became drowsy, then unconscious, and had vomiting and diarrhea.

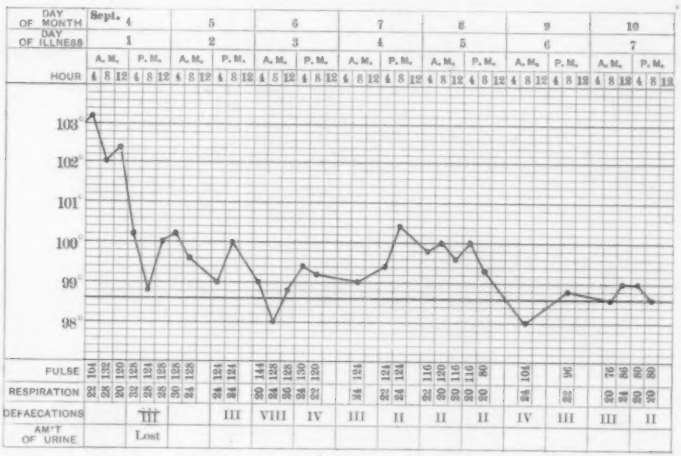

CHART 1.—Case I. Abortive poliomyelitis.

Examination. The patient was somnolent but responded if aroused. There was rigidity of the neck and tenderness of spine. The eyes, ears, nose, were negative except for myosis and tardy reaction of the pupils to light. The abdomen was negative, the spleen was not enlarged. There was a slight Kernig and Babinski of both lower extremities, other reflexes were normal. The highest temperature was 103°.

The case ran the course of an acute dysentery; the stools contained mucus and blood. The urine was negative. Gradually the nervous symptoms became more marked; opisthotonos, delirium, and extreme restlessness developed. A clear fluid escaped under moderate pressure from a lumbar puncture. The number of stools diminish, the temperature and pulse became normal in the course of a week. The symptoms of meningism subsided. The reflexes were never absent at any time.

Although meningism occurs as a symptom of various infectious diseases, especially in children, our original interpretation

of the case did not seem wholly satisfactory, especially after hearing of several cases of poliomyelitis of the gastro-enteritic type which had occurred in the course of the summer. On looking up the patient at his home, a careful examination of his reflexes showed no anomalies. There were no paralyses of any kind. Two ounces of blood were taken from the arms of the patient and subjected to the neutralization test.

This test is made by mixing for many hours, the active virus derived from apes with the serum of an immunized human being, and injecting it intracerebrally in apes. Controls receive in the same manner, virus mixed with the serum from an individual who never had anterior poliomyelitis. The controls contract the disease, while the test cases escape, presumably because of the antibodies present in the immunizing serum. The test in our case showed a positive result. As there was nothing in the patient's previous history pointing to an attack of poliomyelitis, we had doubtless been dealing with a case of the abortive type of this disease.

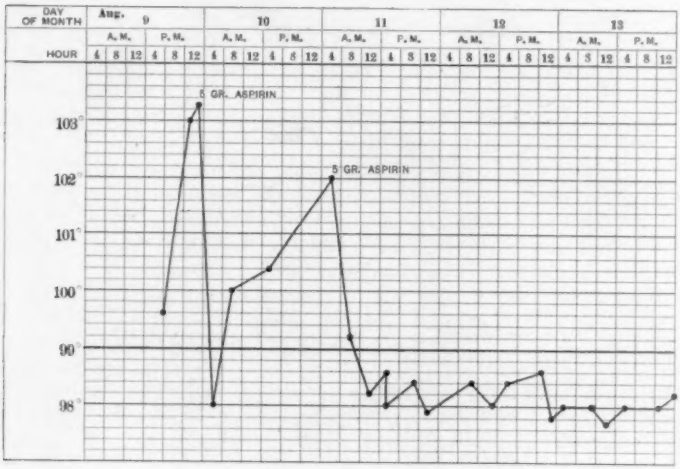

CHART 2.—Case II. Abortive poliomyelitis (meningism).

CASE II.—Since then another case came under my notice and partial observation which allows of the same interpretation. A family with a number of children, travelled through Norway, Sweden, Germany, and France. While in Norway, one of the children had a mild attack of tonsillitis. He subsequently complained of being very tired. Two weeks after leaving Norway, the child complained of headache, which gradually became so

intense that he could not bear his head to be touched, and had to lie down. On the second day, the temperature gradually rose to 103°, pulse, 108. There were no gastro-intestinal symptoms nor cough. The patient gave the impression of being very ill, was extremely irritable, and when he awoke from a restless sleep, complained of severe headache. The pupils were small, but equal, and reacted to light slowly. The patellar reflexes could hardly be elicited, and no Babinski or Kernig phenomena were present. The cremaster and abdominal reflexes were present and equal. There was stiffness of the neck and inability to put the chin on the chest. A tentative diagnosis of cerebrospinal meningitis was made and a doubtful prognosis was given. In the course of the fourth and fifth day, however, the meningitic symptoms gradually disappeared, and the child made an uneventful recovery. Six weeks later, the left patellar reflex was present, the right one absent. There seemed to be a slight paretic condition in the distribution of the right facial nerve. Examinations in the seventh, eighth, and ninth weeks after the attack showed that the patellar reflex of the right leg was gradually becoming normal. This was undoubtedly a case of abortive poliomyelitis.

Case III.—A child, aged three and one-half years, was brought to the German Hospital, August 30, 1911, with a history of fever and diarrhea for a number of days. The mother said the child was unable to stand or walk alone.

Examination. With the exception of a slight rise of temperature, and a number of diarrheic movements, the child, as it lay in bed, did not present anything worthy of note. On placing the child on the floor, however, it could not rise, the feet seemed flabby, the big toes bent under the feet. In a sitting or standing posture, when held by the arm, the trunk reclined to the left side. The pupils and face were normal. The right patellar reflex was diminished, the left normal. Abdominal and cremaster reflexes were present and equal. The electrical reaction of the affected muscles of trunk and extremities were normal. In two weeks after treatment by urotropin, baths, and electricity, the child was able to walk. At the present time there is a distinct limp of the right leg and the patellar reflex on the right side is diminished.

Cases like Case III are not uncommon I presume. On account of the shortness of the febrile stage, they first come under observation when paralysis has set in, and pass on to partial or complete restitution or complete paralysis, according to the severity of the lesions in the anterior horns and surrounding nerve tissue. Frequently, the orthopedist is the one who first sees these cases, as occurred in the following case.

Case IV.—A perfectly healthy but very neurotic girl, aged eight years, complained of headache and dizziness, but insisted on going to school. During the night the child's temperature

rose to 103°; she complained of headache, pain, and heaviness in the lower extremities. On the following morning the mother was thrown into a frenzy, because the child could not sit up or move her legs and had slight difficulty in passing urine. Both patellar reflexes were absent. The temperature was normal on the fourth day, and remained so. Examination showed that the peroneal group of muscles of both limbs were affected, also the posterior tibial muscles, more on the right than on the left side. This case was treated by Dr. J. Teschner with exercises and reëducation. The child never made use of a crutch, nor was a splint applied. She now walks with the characteristic swing and drag of infantile paralysis.

Case V.—This was a healthy, well-developed boy, aged sixteen years, seen in consultation last summer. He had returned from the Adirondacks, with the history of remittent temperatures for three days. The patient complained of heaviness in the lower extremities and pain in the back. An incubation history could not be obtained. Patellar, cremaster, abdominal reflexes were present, equal and slightly exaggerated. There was a distinct Kernig's sign in both lower limbs. The pupillary reaction was normal to light and accommodation. There was slight stiffness of the neck. The spleen was not palpable. There was no bradycardia, the temperature was about 103°. Leukopenia and relative lymphocytosis were present. The Widal reaction was negative. The possibility of typhoid fever or cerebrospinal meningitis was considered. A spinal puncture, however, gave a clear fluid, containing 90 per cent. of lymphocytes. Subsequently, partial flaccid paralysis of the extremities followed. This was a typical case of the spinal form of poliomyelitis.

Case VI.—S. F., a girl, aged about five years, whose mother was tuberculous, was suddenly seized with tonic and clonic convulsions. The patient moved both arms and legs convulsively or held them in tonic flexion; at times the arms and legs were relaxed; then again the legs were held in hyperextension. This was accompanied by twitching of the face, especially on the right side and rolling of the eyes. Severe opisthotonus occurred, which persisted for a number of days. All the reflexes were exaggerated. The pupils were unequal, Kernig's sign was present in both lower extremities, and there was a tache-cerebrale. Her temperature was steadily high, between 104° and 105°. After these irritative symptoms, the patient gradually lapsed into a comatose condition in which she remained for two or three days. During this period, stiffness of the neck and back persisted so that it was impossible to roll the child on her side, as she seemed to resist enforced motion. Spinal puncture was repeatedly made, a clear fluid showing small and large mononuclears escaped under pressure. There was occasional rolling of the hands inward, general tremor, and nystagmus.

The facials seemed at times unequal. The nystagmus gradually ceased, but there remained internal strabismus on the left side. The diagnosis of tubercular meningitis was made, but the tubercle bacillus could not be found in the spinal fluid. Subsequently the child had great difficulty in standing, and the patellar reflexes were much diminished and unequal. Gradually recovery set in. The patient tottered when walking and fell frequently when not guided by the hand. The strabismus persisted for a number of weeks, but gradually disappeared. This was a case of poliomyelitis of the cerebral type.

This case was one of three children belonging to a family that lived in close quarters. During this severe illness of one child the two other children had short accesses of fever, to which not much attention was paid. Both children seemed much prostrated after a few days' illness. It does not seem improbable to me, at the present time, that they too had the short febrile abortive type of poliomyelitis. This may also have been the case with the brother of Case II.

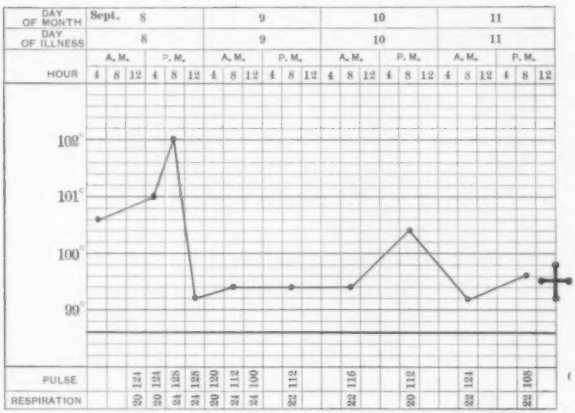

CHART 3.—Case VII. Bulbar and pontine poliomyelitis.

CASE VII.—O. E., male, aged twenty-nine years, entered the German Hospital September 8, 1909. Died September 12. The duration of the whole illness was eleven days. A positive diagnosis was not made, though the existence of an acute poliomyelitis was deemed probable. There was a history of tuberculosis in the family. On September 1 the patient had a slight chill; dull frontal headache and stiffness of neck. He was unable to move the arms at the shoulder-joint; had difficulty in dressing and could hardly

feed himself. He was, however, able to move the elbow and wrist-joints, but muscular action of the arms was weak. No pains.

Examination. The patient was bathed in profuse perspiration, lay helplessly in bed; he was unable to sit up, as he could not support himself on his arms or hands. The eye muscles were normal and the pupils reacted normally to light and accommodation. There was stiffness of the neck, inability to move the head from side to side, or flex it on the chest, and moderate rigidity of the spine. No hyperesthesias. There was complete loss of motion at both shoulder-joints, yet extension of the forearms was accomplished promptly. The hands and fingers were readily moved. Considerable muscle weakness existed. There were no paresthesias or hyperesthesias, no special points of tenderness along the nerve trunks. The knee-jerks were not obtainable; Kernig, Babinski, and Romberg signs were absent, and there was no loss of function or weakness of the leg muscles. No disturbance in sensation or electrical reaction of either upper or lower extremities occurred. Lumbar puncture was dry. The patient acted peculiarly, screamed during sleep, talked irrationally, wanted to get out of bed. On September 11 he was slightly delirious, and had some difficulty with speech and swallowing. The patient died suddenly during the night. The autopsy showed hemorrhages in the upper part of cord and pons. This was a case of the bulbar and pontine poliomyelitis of Oppenheim and Erb.

Case VIII.—Female child, aged two and one-half years. Bottle-fed, well nourished. In September, 1908, she had an attack of enteritis with fever. The child seemed very restless and nervous; complained of headache, and was suddenly seized with general convulsions which lasted for two days and nights. Chloroform was constantly administered. There was convulsive twitching of the face and tongue, with inability to swallow. The temperatures was up to 105°. The pupils were contracted. Bilateral Kernig's sign was present. The abdominal and patellar reflexes were not exaggerated, and there was no foot clonus. Trousseau's sign was present. Spinal puncture gave a perfectly clear fluid. At no time was there stiffness of the neck. The irritative symptoms persisted about a week, then the upper and lower extremities gradually relaxed and the pupils became dilated. The patellar reflexes disappeared. It was observed that the child did not take notice of objects placed in front of it, or held before its eyes; an examination of eye grounds by Dr. Koller showed hyperemia of the retinal vessels, but no optic neuritis. The child had a flaccid paralysis of the lower extremities, and was unable to stand. The hands were held in flexion. No reactions of degeneration were present. As there was an extended epidemic of cerebrospinal meningitis at the time, the question as to whether we were dealing with this infection was also favorably considered. In view of the

fact, that opisthotonus was never present, that the spinal fluid was clear, and that the attack was ushered in by a severe enteritis which ran parallel with the nervous symptoms, I am inclined to uphold my diagnosis of encephalitis, in which I was supported by several competent observers. I would group this case with the encephalitic type of poliomyelitis, in the light of our present knowledge of this disease.

Observers of the meningeal and cerebral forms of poliomyelitis like Wickman[8] and others go into a very careful differential diagnosis between this type of poliomyelitis and cerebrospinal meningitis. In exhaustive treatises[9] of epidemic serebrospinal meningitis, however, the differential diagnosis of the two diseases is not even considered. From this I infer that the cerebral type of poliomyelitis has hitherto often passed unrecognized.

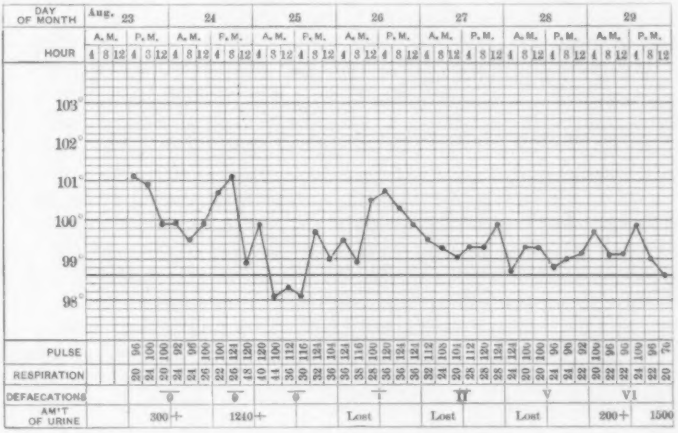

CHART 4.—Cerebral type of poliomyelitis.

CASE IX.—The following case is of interest in this connection. A native German, male, was brought to the hospital in a comatose condition on September, 1911. There was no history available excepting the fact, that he had come from the Catskills, where a few cases of infantile paralysis had occurred. Soon the patient was seized with tonic and clonic convulsions of the right side of the face, arm, and leg. Between the attacks the arms were held in fixed flexion. The right pupil was larger than the left. All

[8] Loc. cit.

[9] Goeppert Epidemic Cerebrospinal Meningitis (Genickstarre) Ergebnisse der Inneren Medizin und Kinderheilkunde, 1909, Band iv, p. 165.

the reflexes were exaggerated and were stronger on the right than on the left side. Foot clonus, Oppenheim and Babinski reflexes, were present on both sides. Abdominal and cremaster reflexes were intact. On stroking the plantar side of the right foot with the hammer a very strong contraction of the vastus of the right thigh was produced, which on repeated manipulation became tetanic. There was slight stiffness of the neck and complete aphasia. His temperature was up to 103°. Leukocytosis, 12,000.

The convulsions ceased, then started in again. They were controlled by the administration of bromides with the stomach tube. There was a strong probability of this being a case of polioencephalitis, poliomyelitis of the encephalitic type, first described by Strümpell, with the identical symptoms, especially in consideration of the etiological factor. Careful subsequent examinations, however, showed a very distinct scar on the penis, the contours of the pupils were unequal (anechoria syphilitica), the Wassermann reaction was strongly positive. The spinal fluid showed an excess of lymphocytes, and the Nagouchi reaction was positive. After recovering from the coma, the patient gave a complete history of syphilitic infection. The patient is still at the Hospital, convalescent, and improving daily.

From the sketch of the histories given above, it becomes apparent that the diagnosis of this disease in the acute stage is, to say the least difficult, and under certain circumstances impossible. Some experienced observers state, that the four cardinal symptoms presented are, subjective pain, hyperesthesia, perfuse perspiration, and leukopenia. Each one or all of these symptoms may be absent or present. Leukopenia, which is mentioned as a constant symptom is by no means always present. Moderate leukocytosis may be found due to the accompanying catarrhal affection of the respiratory tract or intestinal canal. As the early stages of measles and the great majority of the cases of typhoid fever also show leukopenia, the symptoms is only of value, if it occurs at all, in connection with the whole clinical picture. If the above-mentioned symptoms fail or are inconstant, how then is it possible to obtain the *first clue* to the existence of this disease and to differentiate it from other febrile conditions? I strongly suspect that this is impossible in certain elementary or transitory attacks of a mild character. In most cases, however, we will be rewarded by a most painstaking search for symptoms pointing to an irritation of the cerebrospinal system. It is unnecessary here, to go into a detailed analysis of the well-known signs of the presence and absence, exaggeration and diminution, and variability of the reflexes of the eyes and the muscular system which belongs to a complete neurological examination. The variations exhibited in the reflexes is due in great part to the location and to the irritative or destructive nature of the inflammatory process in the neurones. Recent

pathological study has furthermore shown, that it is not only the meninges, the anterior horns of the cord, the cortex of the brain, and the central nuclei which may be affected, but also the posterior horns, in fact, any part of the brain and cord may be implicated. The variability of the nervous symptoms is in itself if not characteristic, very suggestive. The taking of a complete nerve status at each visit will soon uncover the mutability of the reflexes, the motor and sensory disturbances. The first point to be looked to is, therefore, to discover, if possible, any sign of meningeal or cerebrospinal irritation. John Lovett Morse[10] has recently corroborated the observations of Brudzinski, who first pointed out two distinct phenomena which are not present in healthy children, or in diseases other than those of the brain and cord. These are: (1) The contralateral reflexes of the legs, and (2) the neck sign. The contralateral reflex consists in the concomitant reflex of the leg on one side, when passive flexion of the leg on the other side is made. There are some variations of this sign. Brudzinski's neck sign is present in meningitis when passive flexion of the neck forward causes flexion of the legs at the hips and knees, and a marked flexion of the legs on the pelvis. Various writers have attached great importance to these signs in the diagnosis of meningitis. Further study must show, whether these reflexes are present in the meningeal forms of poliomyelitis. In a former paper[11] I dwelt upon the importance of spinal puncture in the diagnosis and treatment of various diseases in young children. At that time I hardly suspected the significance of the procedure in the diagnosis of the subject now under discussion; although it will take a far more extended experience than we now possess, and perhaps the devising of new methods of investigation to differentiate specifically the spinal fluid found in the various infections. In a careful analysis of over 500 specimens Lucas[12] tabulates the findings—macro- and microscopic of the spinal fluids of the various infections of the brain and cord in young children.

The cases under consideration may be divided into two large groups: (1) Cases with a predominance of polynuclears; (2) cases with a predominance of mononuclears or lymphocytes. The first group includes cerebrospinal meningitis as well as influenzal, pneumococcus, and streptococcus meningitis. In addition to this the characteristic organism is found in the spinal fluid in each instance. The second group, with predominance of lymphocytes, includes meningism, encephalitis, syphilis of the central nervous system, tubercular meningitis, and poliomyelitis. There is no reference to spinal puncture in tetany in the literature.

Let us attempt to differentiate the diseases belonging to the

[10] Archiv. Pediatrics, 1910, xxvii, No. 8, 561.
[11] Amer. Jour. Med. Sci., 1910, cxxxix, 542.
[12] Amer. Jour. Diseases of Children, March, 1911, p. 230.

group with lymphocytosis in the spinal fluid. In syphilis of the central nervous system, which is comparatively rare in children, the spinal fluid may give the Wassermann reaction. It also gives the positive Noguchi butyric-acid test. In tuberculous meningitis the Noguchi reaction is negative; the tubercle bacillus is present in nearly 100 per cent. of the cases in the spinal fluid, as was shown by Hemenway. As far as encephalitis is concerned, a number of these cases may be of a parasyphilitic nature and give the Wassemann reaction, or they belong to the cerebral or encephalitic forms of poliomyelitis. It is not unlikely that some cases cannot at present be classified.

The subject of meningism[13] presents serious difficulties and needs further study. The cases belonging to this group must be considered in connection with the underlying infection; the cytology and bacterial findings will vary according to the early or late stage of the disease.

As to poliomyelitis, Flexner and Lewis,[14] Gay and Lucas,[15] and others, have studied the spinal fluid in monkeys and in young children. The result of these studies can best be illustrated by the precise diagnosis of an obscure case from the microscopic examination of the spinal fluid. This was the case of Dr. Frissell: The patient was a young adult in whom all the reflexes were exaggerated, the Kernig sign and Babinski reflex negative, and the neck a trifle stiff. The Widal, and blood culture for the Bacillus typhosus were negative. Leukocytes, 12,000. Physical examination was negative. On the fourth day of the disease, the first lumbar puncture was made. Flexner reported that the fluid was turbid, the sediment consisted of a small number of large lymphocytes, a considerable number of small lymphocytes, and very few polynuclears, and that the clear supernatant fluid gave the Noguchi reaction. The condition of the fluid resembled that seen in inoculated monkeys, just before the onset of the paralysis. On the fifth day of the disease, the reflexes nearly disappeared and there were indications of paralyses of the lower limbs and abdomen. A second spinal puncture showed that the fluid was clear and still contained an excess of lymphocytes. The Noguchi reaction was not so strong. This second fluid resembled that of monkeys after paralysis had set in. The diagnosis and prognosis of this case were made by the most advanced clinical and laboratory investigations.

Nothing is more commendable in clinical medicine than to lay special emphasis upon the thorough observation of the case in hand in all its various clinical aspects. All our methods of observation, both at the bedside and in the laboratory, should be brought to bear on the determination of the given case, just as in criminal

[13] Johns Hopkins Hosp. Rep., 1904, xii, 379, and Richard Stein, Ibid.
[14] Archives of Pediatrics, 1910, xxvii, No. 2, p. 93.
[15] Archiv. of Int. Med., 1910, cxxix, 542.

law the jury sifts the evidence of the witnesses and takes note of the impression made by the prisoner. The finger-print method of identification seems at present, however, destined to become the scientific test which will outweigh the sum of all the circumstantial evidence; so in our own field the special diagnosis of disease by laboratory methods is advancing in leaps and bounds. Bacteriological, serological, biochemical tests have opened a great vista in the recognition of disease by scientific methods. In acute poliomyelitis we are dealing with a disease, sudden in onset, short in its course, with symptoms obscure and elusive. Although the attempt to diagnosticate it by the serological method has so far been futile, let us hope that its recognition by a simple scientific laboratory test—not to speak of its prevention, cure, and extermination—will be effected in the not too distant future.

A CLINICAL CONTRIBUTION TO OUR KNOWLEDGE OF POLIOMYELITIS WITH CORTICAL INVOLVEMENT.[1]

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It can hardly be supposed that the more intensive study of epidemics of poliomyelitis of recent years accounts for the bizarre clinical manifestations of this disorder which are now of frequent report. The situation would seem of more rational explanation on the ground that the isolated atypical forms of the disease were overlooked or disregarded in the past. The bulbar and pontine types are now common and well defined, but the cerebral and especially cortical involvements are still subjects of debate despite the classic and well-known contentions of Strümpell and his school on the subject.

The existence of a true encephalitic type of poliomyelitis is difficult to differentiate clinically as well as pathologically. In the first place, a spastic palsy is not definite proof of cortical or encephalitic involvement, and secondly, when the autopsy has shown cortical or meningeal alteration the findings have been susceptible of explanation upon other causative grounds. It is not sufficient to state that an encephalitic type must show a cortical lesion and present evidences of notable meningeal irritation, resulting in hemiplegia, which later becomes spastic, but the character, onset, and clinical course of such cases must be a matter

[1] Read before the New York Neurological Society, October 3, 1911.

of careful inquiry before a diagnosis of cortical involvement is possible. Opponents of Strümpell's views no doubt hold too rigidly to a true clinical picture of poliomyelitis itself to admit of many of the bizarre symptoms which must obtrude themselves into the make-up of the clinical syndrome of the cerebral type of the disease. A mere report of a sequential palsy, flaccid or spastic, is susceptible of varied interpretations in the individual case. Many times the apparently associated spastic-flaccid states reported are but instances of flaccid palsy with contractures in normal groups of muscles. Taking the matter by and large, there can be no doubt that the few and infrequent case reports of Strümpell's type show that polio-encephalitis in its typical form is seldom encountered in epidemics of acute poliomyelitis and perhaps occurs only in certain epidemics. Thus, in the Report of the Collective Investigation Committee on the New York Epidemic of 1907, on Epidemic Poliomyelitis,[2] it is stated that "while the committee do not doubt the rare occurrence of the cerebral (cortical) type of the disease, no undoubted case of this form was reported and in not a single family was it stated that one child was affected with the acute spinal form of the disease and another with the acute cerebral form. Strümpell's hypothesis of the analogy between polio-encephalitis and poliomyelitis is still in need of strict clinical and anatomical proof. The cases previously reported by Moebus, Pasteur, Bucelli, and the doubtful ones of Hoffman and Strümpell are the only cases that offer any support to Strümpell's contention."

Even when we consider the less debatable form, the meningeal type, the subject is not less complicated and is equally difficult to demonstrate in the epidemic disease. It is very difficult to exclude the other kinds of meningitis, not alone epidemic cerebro-spinal meningitis, but also tubercular meningitis.[3] Meningeal irritation in acute poliomyelitis is not necessarily an initial symptom, but may develop after some days of malaise, etc. Epidemic cerebro-spinal meningitis may occur at the same time and place as epidemic poliomyelitis and must, therefore, be excluded. Even a very wide questionare study of epidemics by Zappert[4] failed to establish a complete picture of any meningeal form. He cites 4 cases supplied by this correspondence study. In one family a child, aged eight years, died of meningitis, while one, aged four years, developed a mild case of monoplegia (left leg), and one, aged three years, a paraplegia following what was apparently a severe gastro-enteritis. In another household attack, one child developed a

[2] Nervous and Mental Disease Monograph Series, 1910, No. 6, p. 53.

[3] Koplik, The Cerebral Forms of Poliomyelitis and their Diagnosis from Forms of Meningitis, Amer. Jour. Med. Sci., June, 1911, No. 471, p. 788.

[4] Epidemic of Acute Anterior Poliomyelitis, Jahrb. f. Kinderheilkunde, 1910, Band lxxii, S. 107.

paraplegia, its sister a meningitis which ended in recovery. In a third family, two children developed mild transitory meningeal symptoms. Of more interest was a fourth family episode. One child developed meningeal symptoms from which it recovered, but a monoparesis remained (right leg dragged in walking). A brother also developed a meningeal syndrome and temporary right hemiplegia resulted.

While most of us are, therefore, prone to admit, both from clinical as well as pathological evidence, that meningitis may *co-exist* with acute epidemic poliomyelitis in the same subject, an *exclusively* meningeal type appears to be doubtful. If one looks for proof of encephalitis in epidemic poliomyelitis by the presence of cerebral diplegia alone, classic instances of the association are even more rare than the pure meningeal forms just considered. Most writers upon the subject have heretofore confined themselves to this rather rigid test of a pure encephalitic type. If, however, the latitude of inquiry is enlarged to permit of a hemiplegic type also, as well as certain cerebral symptoms known to be cerebral or cortical in origin (such as choreas, athetoses, aphasia, and convulsions), data are not so difficult of attainment. Thus, in the Swedish epidemic of 1905, Wickman saw no cases of spastic hemiplegia, but other symptoms were recorded which pointed to a cerebral localization. Thus, a boy, aged five years, had the usual febrile onset with violent spasms. He could not speak or leave his bed. The spine was somewhat rigid and tender. The head was bent backward, the hands and arms were weak. He was unable to stand without aid. In about a week he had improved greatly, but still dragged the left leg. The knee-jerks were heightened. In this case the motor aphasia certainly showed a cerebral lesion. A medullary location could be excluded. The heightened knee-jerks also spoke for cerebral localization. The usual atrophy of poliomyelitis was absent. The presence of meningeal symptoms suggested a meningo-encephalitis. The lesion must have been trivial, as the patient entirely recovered. Lunggren also saw a patient with motor aphasia in this same epidemic. Bohmann also saw two brothers, one of whom had typical poliomyelitis. The other, aged one year, had a severe right unilateral convulsion followed by paralysis of the whole right side. The child quickly recovered. The case was an unmistakable cerebral and probably a meningo-encephalitic form of epidemic poliomyelitis. However, no author has apparently seen the full development of the typical syndrome of poliomyelitis associated with spastic paralysis, which further illustrates the necessity of broadening the clinical picture if we are to gain records of cortical types of poliomyelitis. In autopsies, however, acute encephalitic and cortical foci in typical epidemic poliomyelitis are of rather frequent report. Redlich found hyperemia of certain cortical vessels and round-celled infil-

tration in the basal ganglion. Wickman has found the same round-celled infiltration in the cortex of both hemispheres. They were numerous and disseminated. The same lesion was also present in the central ganglia. In another case cited by Wickman no infiltration was present, but there were areas of hyperemia and slight transudation with abnormal richness in nuclei. It must be said, however, in nearly all cases the lesions were of microscopic dimensions. In autopsies of adults the coincidence of old poliomyelitic and encephalitic lesions have also been recognized. Clinical data in this connection, while few in number, are of great interest. Flaccid and spastic paralyses have been seen side by side in children of the same family by Mobius, Medin (4 cases), Pasteur, Bucelli, and Hoffmann. In 1899 Williams reported an association of flaccid and spastic paralysis in the same patient. The child was first seen when eleven years old. When only five years she had headache and convulsions followed by right hemiplegia and asphasia. In a little over a year she recovered or at least improved. He found spastic paralysis of the right arm with flexion contracture. This arm was stronger than the left, probably from its constant choreic twitchings. The right leg was smaller than the left. The knee-jerks were normal. Electric reactions for arm and leg muscles were normal save for the peronei, which showed reaction of degeneration. Analogous cases have been reported by Neurath, Calabuse, Negro, Oppenheim, Marie, and Rossi.

Wickman saw an analogous case as far back as 1899. A girl, aged eleven years, showed evidence of an encephalitis in infancy, before she had learned to walk. The legs were weak for a year, but recovered enough so the child could creep about. When the little patient was first seen, at the age of eleven, there was a high degree of paresis of the lower extremities more marked on the left side. She was unable to stand or walk unaided. When assisted to walk the gait was paralytic, not spastic, but flexing one knee brought out resistance. The muscles of legs were atrophic. There was no reaction of degeneration, but the electric reactions were weak. Intelligence was depressed. Left knee-jerk abolished, right increased. Complete analysis of muscular and tendon phenomena points to an association of cerebral and spinal lesions.

One may summarize the cerebral involvement in poliomyelitis on its anatomic-pathological side as follows: There may be a disseminated encephalitis affecting not only the white substance of the pons and crura cerebri, but also the gray matter of the cortex and regions of the central ganglia. The process, in some instances, would seem to originate in the pia, thus furnishing an important document for the fact that in the epidemic disease the palsy or injury may be a cerebral instead of a spinal one. The random and often unilateral character of the lesion in the cortex is a parallel picture to the pure spinal types. The rarity of the cerebral type

is accounted for by Wickman on the ground that epidemic poliomyelitis is an ascending disease and, therefore, must tend to involve the bulb before it reaches the brain. However, it must be said that in experimentally induced poliomyelitis in apes, no encephalitis can be induced, thus seeming to contradict this theory of extension of the virus up the cord by means of the lymph spaces. It is, however, impossible to inoculate the ape's cortex or subdural spaces directly. We are, therefore, forced to conclude that the lumbosacral cord is most predisposed and the cortex least so. As before mentioned, a simple spastic state cannot necessarily be ascribed to a cerebral, or at least, a cortical involvement, as not infrequently the bulb shows such extreme involvement that the pyramidal tract at this juncture is often seriously damaged.

Thus, Müller has seen 4 cases of spastic paralysis none of which could be explained by a polioencephalitis of the cortex solely. One of his cases, a child, aged seventeen months, suddenly developed a spastic hemiparesis after a febrile attack. This was explained by a lesion of the pyramidal tracts within the pons as shown by the crossed peripheral facial palsy. In another case there was a spastic state in one lower extremity and a flaccid palsy in the other. In still a third, there was a hypotonic paresis of the leg and a hypertonic paresis of the arm. There can be little doubt that in the majority of cases with spastic palsies the lesions inducing the same are at the bulbo-spinal level rather than cortical. A very few cases, however, are on record, as reported by Müller and Förster, where a disseminated encephalitis must have been the predominant lesion. I am not aware, however, that any one has reported an exclusive encephalitic lesion in a case of undoubted clinical polioencephalitis. In other words, the clinical and pathological report have not yet coincided in the one case. If the spastic state is, therefore, not a criterion of cortical involvement and can only be considered presumptive evidence, what other facts must we include to make the lesion definitely a clinical as well as a pathological fact? Obviously, the presence of chorea or athetotic movement cannot be a part of the positive criteria as lesions giving rise to these symptoms may be, and indeed are, most frequently subcortical in origin. True motor aphasia, enduring mental enfeeblement such as idiocy and genuine grand mal epilepsy would seem to make the diagnosis of cortical involvement positive. Of all these phenomena genuine and enduring epilepsy might be definitely considered to make the diagnosis of a cortical lesion most certain. I have, therefore, such a case report.

F. S., aged eighteen years. He suffered greatly from indigestion and constipation as a baby. When aged two years, he had one teething convulsion. When aged nine years, after an accidental fall in the water, he had a single convulsion on attempts at resuscitation. When aged fourteen years, he had a typical

attack of poliomyelitis as follows: The day before the paralysis set in he had fever, vomiting, prostration, and a convulsion; the next morning he attempted to walk across the room; he had taken but a few steps when he fell and suffered a complete paralysis of all voluntary muscles. The patient suffered a good deal from pain in the muscles of the lower extremities. He slept poorly, but was not delirious. He remained acutely ill for two or three days, could not sit up for several days and was able to stand only with crutches after two or three weeks. The paralysis improved slowly but steadily from above downward. He used crutches to get about for six months. When I first saw the patient one year ago for his grand mal epilepsy which has occurred regularly two or three times every three or four months since the attack of poliomyelitis, he showed a steppage gait, more marked in the right foot. There was moderate palsy and atrophy in the tibials and peronei of the legs. There were reactions of degeneration and some shortening of both postici. The whole condition was most marked in the right leg. The knee-jerks were barely present, being difficult of attainment on the right. There was a normal big toe reflex.

Here, then, is a case of poliomyelitis and epilepsy associated in the same case. Can it be considered a mere coincidence? It may be urged that the occurrence of a convulsion at dentition would argue against the case being one of epilepsy induced by cortical injury in poliomyelitis at fourteen years of age. When one bears in mind, however, the fewness of eclamptic convulsions in infancy that terminate in epilepsy, not more than 2 per cent., the importance of the attack at dentition is of little moment. Again, the boy having a convulsion the result of asphyxiation at eight or nine, after a prolonged submersion (ten minutes), but shows the predisposition to convulsions upon a sufficient cause presenting itself. The ordinary frequency with which spasm and convulsions are encountered at the outset of all infectious fevers in infancy ought to cause one to expect the same phenomenon more frequently in poliomyelitis, a disease of sudden febrile onset. Even supposing that the epilepsy in our case was but one of this character, the few cases of epilepsy or eclamptic attacks examined at death, the sequel to infective fevers, bear no distinct pathology of the cerebral cortex not seen in the reported cases of cortical involvement in poliomyelitis. I am fully aware that these and other reasons for looking askance on my case will occur to many; but before I had unearthed the specific fever (poliomyelitis) at fourteen years of age I had given my opinion that whatever the febrile movement was or had been caused by, his disease as we now see it was induced at that time and was dependent upon that particular infection. The usual mental stigma of a pure idiopathic epilepsy is not here in evidence. The disease

in its classic form appeared promptly and has endured since the severe attack of poliomyelitis. Indeed, the first grand mal attack with bladder incontinence and tongue biting came on *pari passu* with the fever and prostration of the poliomyelitis at its inception.

It would seem that we have strong evidence that this patient suffered a diffuse cortical injury at the onset of the poliomyelitis, leaving organic changes in the cortex producing his epilepsy. I can find no similar case on record. The epilepsy in my case has responded illy to the usual hygienic plan of treatment so beneficial in pure idiopathics. The case is placed on record at this time as a clinical contribution to the possible occurrence of cortical injury in a case of otherwise classic poliomyelitis. The residue of the cortical involvement is shown in an enduring epilepsy, a fairly well-proved cortical disease.

While it must be admitted that general or focal convulsions do occur at the onset of polioencephalomyelitis these convulsions or phenomena are for the most part epileptiform in character, lacking the complete picture of genuine epileptic fits, or, they are confined to the acute onset of the epidemic disease or, at least, to the very few days immediately following the attack. There are no cases I have been able to review in the literature in which the convulsions continued as genuine epilepsy in after years such as I am here recording. It is, of course, quite conceivable that this outcome might be expected to occur, as in my case, but it has not come to my notice. The above explanation and disposition holds good for such cases as those of Müller, Zappert, D'Alloco, and Brorström.

It is true that no two nervous disorders are mutually exclusive and in the natural order of events one might expect a certain but small number of epileptics who also had suffered a poliomyelitis. I have seen or known of but one such case in an experience covering several thousand examinations. It may be of interest to cite briefly the details of this one case:

L. L., male, aged thirty-eight years, suffered from a mild attack of poliomyelitis when aged eight years, which affected the whole right extremity. There is now a wasting of the whole right leg from the hip downward and some shortening has resulted. Slight talipes equinovarus and contractures in tibialis posticus of this same side is in evidence. Left knee-jerk is nearly absent while the right appears nearly normal. Achilles jerk is active on the left side and absent on the right. There is a normal big toe reflex. Reaction of degeneration is present in tibiales and peronei. There was a neurotic family history and alcoholism in the father. Patient's first seizure occurred at eighteen. The attacks were grand mal in type, preceded by no aura and were mostly nocturnal. At first the attacks occurred every two or three months; now they are

of weekly onset. There is considerable mental deterioration at present.

The case was admitted to my service at Craig Colony several years ago and is still there, a confirmed epileptic. I am indebted to Dr. Shanahan, Superintendent of the Colony, for the case notes here given.

NOTE.—Since this article was written, Richard Stern[5] has published a study upon the future of poliomyelitic children, and has found two cases of epilepsy developing several years after the initial disease. His material embraces 107 cases of poliomyelitis. This author suggests that so long an interval between the poliomyelitis and the subsequent epilepsy took place that suspicion is justified that the affections were coincidental. No data whatever are added. The cases are identical with my case, L. L., in which epilepsy was sequent to poliomyelitis contracted in infancy and in which the comitial disease appeared ten years after the onset of the acute poliomyelitis.

THE VALUE OF NAUHEIM BATHS IN NEPHRITIS WITH HIGH BLOOD PRESSURE.

BY E. AVERY NEWTON, M.D.,
BAD NAUHEIM, GERMANY.

DURING 1905 and 1906, while associated with the late Dr. Heineman in his work at Nauheim, I paid particular attention to the blood pressures of patients before, during, and after taking the baths, and found in cases of high blood pressure (170 mm. Hg. and over) resulting from kidney complications, that the warm saline baths had a more beneficial effect than the cooler baths containing carbonic acid gas.

Up until that time I had always shared the general belief that Nauheim baths were absolutely contraindicated in all cases of marked kidney disease, and in the literature on the use of the Nauheim treatment which I have consulted, kidney disease, when mentioned at all, is regarded as a contraindication to the baths.

The first thing mentioned by Heineman[1] under contraindications to the Nauheim treatment is chronic Bright's disease. Richard Greene[2] says: "After numerous trials, carefully watched and carefully conducted, Schott found that almost any form of heart disease was benefited by the treatment he elaborated. I have said

[5] Jahrb. f. Psychiatrie, etc., 1911, No. 182.
[1] Medical Record, June 22, 1901.
[2] The Schott Treatment for Chronic Heart Diseases.

almost, because it would manifestly be unsuitable for thoracic aneurysm, and much care would have to be observed in treating cases of extensive aortic insufficiency. Also in cases complicated by arterial capillary fibrosis, and small granular kidney." While Groedel[3] as recently as 1910 expressed the following opinion: "Bei patienten mit ungewöhnlich hohem blutdruck, 190 mm. Hg. und mehr, haben wir zuweilen, gerade infolge der verminderung des blutdruckes, schwindelanfälle gesehen. Meist sind dies patienten mit gleichzeitiger nierenaffektion. Wir warnen deshalb davor, solche patienten zur kur nach Nauheim zu schicken, desgleichen patienten mit sehr grossen aneurysmen."[4]

These observers, whose wide experience with the Nauheim treatmend lends weight to their statements, voice the general belief on this subject. I have found that nearly all physicians are of the opinion that patients with marked kidney lesions are unsuited to the Nauheim baths. My own experience, however, is at variance with this view, for not only have I failed to find a case of nephritis that has in any way been harmed by taking the baths at the proper temperature, but on the contrary, all of my cases have improved. We know that cold bathing, generally speaking, is harmful in such cases, and the baths as usually employed in Nauheim at a temperature of from 83° to 90° F. (28° to 32° C.) are comparatively cool baths. This fact may explain, in part at least, why patients with kidney disease have, as a rule, actually been made worse by the taking of such baths.

In my work at Nauheim during the past three years I have observed a number of cases referred for the cure, in whom various grades of nephritis were present. All of these cases, without exception, were improved by the use of the mild, warm, saline baths given at or near 95° F. (35° C.). Many of these cases have arrived with systolic blood pressures ranging frome 170 to as high as 230, and in all of them the blood pressure has been reduced and the kidney condition improved, in some cases albumin and casts have entirely disappeared. Some of these patients have returned once, and others twice, and in these cases I have found that the benefits derived from the first cure have been lasting, some of them showing no albumin and casts on their return a year later, while in others the albumin and casts, though present, were found in less amounts than on the first visit, and the blood pressure had remained lower.

The beneficial influence on high blood pressure and albuminuria which I have observed following the use of suitable Nauheim baths is best shown by the following cases:

[3] Bad Nauheim, 1910, p. 118.

[4] With patients who have unusually high blood pressure, 190 mm. Hg. and more, we have noticed occasional dizzy spells on account of the diminution of the blood pressure. Most of these are patients who have kidney trouble at the same time. We, therefore, warn against sending such patients for a cure to Nauheim, also patients with large aneurysms.

CASE I.—M. D., aged thirty-seven years, married twelve years; one child living, aged three years; two miscarriages. Severe headaches since miscarriage in 1904; eclampsia at birth of last child, an eight months' gestation. She was totally blind following this for two months, when she began to see rays of light and could tell light and dark. Her eyes have slowly improved, until she now has 1.5 vision. While looking at an object it usually changes color. Nearly everything looks red, changing to yellow or purple.

When this patient arrived at Nauheim, July 19, 1909, her blood pressure was 160, pulse 100, and she was very nervous. The urine contained a trace of albumin, but no casts. As her time was limited, she was able to take only eighteen warm saline baths. On her departure her blood pressure was 130, pulse 80; she was much less nervous, and her headaches were less severe.

She returned on May 17, 1910, feeling much better than on the previous year. She now had almost none of the high pressure and headaches, unless she overdid. Her eyesight was much improved, but far from normal. The blood pressure on arrival was 145, pulse 80. The urine contained no albumin nor casts. While here she was given twenty-three warm saline baths, to the last six of which mother lye was added. She was then sent to Switzerland to rest four or five weeks. She returned to Nauheim on August 4, and was then given eighteen warm, saline baths, to which, after the sixth bath, mother lye was added.

A note taken June 28, 1910, states: She feels much relaxed from the baths, vision steadily improving; every few days she sees things about her clothes and room which she did not notice before. The improvement in detail and color sight is most marked. In 1909 blue, gray, and lavender were all the same color to her, and usually could not be distinguished from white. While in a jewelry store a little over a week ago she looked at two pieces of jewelry—one lapis-lazuli, the other amethyst, one gold mounted, the other silver. Both looked alike to her then. Two days ago she was looking at them and the colors were quite distinct. She says that her ability to distinguish colors has improved remarkably the last few weeks since she has been taking the cure at Nauheim.

In January of 1911 I saw her in San Francisco. She was able to read, write, and play cards. I went with her to the ophthalmologist, who had made repeated examinations of her eyes since her attack in 1906. He told me that for a year or more before her first visit to Nauheim her eyes had not improved, and that he was much surprised at the marked improvement after her return from there, and that there had been even more improvement since the last cure than there had been after the first course of treatment at Nauheim.

She returned to Nauheim for treatment on July 20, 1911. Her blood pressure was 135, pulse 70. Her urine was free of albumin, and there were no casts. She had had practically no headaches

except very slight ones after indiscretions in diet. While here she was given sixteen termal or brine baths. The blood pressure and pulse remained the same.

CASE II.—M. L., aged fifty-eight years. He arrived at Nauheim July 27, 1909. He had been first sent to Nauheim in 1901 on account of cardiac dilatation and endocarditis. At that time he was only able to take a few baths, and had to use a wheel chair constantly. A week after he left Nauheim he began to feel much better, and gained constantly. He returned in 1902, took the first and second cure, was much improved, and continued to feel well several years thereafter.

Three years ago, on awakening, he found his right leg cold. When examined by a physician a thrombophlebitis was found. Later he was sent to Nauheim, where examination showed that his heart was dilated, the left border extended one and one-half inches beyond the left midclavicular line; the first sound was weak and diffuse, the second sound normal; his blood pressure was 160, and his pulse 104. He was given sixteen warm saline baths and four termal, sprudal baths. His blood pressure was gradually reduced to 130, and his pulse came down to 80.

His urine on arrival showed albumin, 0.014 per cent., and hyaline and granular casts. One week later the albumin was 0.012 per cent., there were hyaline, but no granular casts. Ten days later there was 0.003 per cent. of albumin, a very few hyaline, and no granular casts. On August 25, eight days later, the quantity of albumin was too small to estimate quantitatively, and there were no casts.

CASE III.—An attorney, aged fifty-eight years, arrived at Nauheim July 22, 1910. He used tobacco and alcohol moderately; had always slept well until a short time before, and when first seen was sleeping poorly. His appetite was ravenous, bowels very constipated. In the spring of 1897 he had a severe attack of "la grippe," and was much prostrated. During his convalescence he had a relapse, with an attack of acute Brights' disease, which lasted for several weeks. He had no further trouble until the spring of 1910, when he noticed marked shortness of breath on going upstairs or going up an incline. This became progressively worse. He had an attack of heart failure early in May, 1910, after which he was unable to work or exert himself in anyway. He came to Nauheim to avoid petty annoyances, more fancied than real. His son, who accompanied him, had to watch him closely, as most of the time he was confused. Had to break the journey twice between Antwerp and Nauheim because of fatigue.

Examination showed that the heart was enlarged; the left border extended two inches outside of the midclavicular line, the first sound was about normal, but the aortic second was markedly accentuated. The pulse was irregular, and as nearly as could be

estimated had a rate of 92. The blood pressure averaged 180, with occasional beats up to 190. While at Nauheim the patient was given twenty-six warm, termal baths. Improvement was marked in the character of the pulse, and quality of the heart sounds. The heart diminished in size, until on his departure, the left border of cardiac dulness was just outside of the midclavicular line. In this case the blood pressure did not go below 180. The pulse finally became regular and averaged about 70.

The urine examination on arrival showed albumin, 0.031 per cent., hyaline and granular casts; sugar, 0.52 per cent. One week later the albumin was 0.009 per cent., there were a few hyaline and granular casts, but no sugar. One week later the albumin was down to 0.008 per cent., a few hyaline but no granular casts were present, and sugar was still absent. The last examination made on August 25, 1910, showed only 0.006 per cent. of albumin, a very few hyaline casts, and no sugar.

Case IV.—A banker, aged sixty-nine years, married, arrived in Nauheim August 2, 1909. He had always been well until 1895, when he had a severe attack of appendicitis; made a slow recovery, and came to Europe where he lived for five years. After this he returned to business and felt able to do more work than usual. In 1907 he noticed some indigestion, and later discomfort after eating. He was examined and found to have a high blood pressure. He spent the winter and spring of 1908 in Egypt and Italy, returned to his home in the United States in April, and felt much better. The following summer he came to Nauheim.

Examination at that time showed his heart to be enlarged, the left border extending about one inch outside of midclavicular line; the first sound weak and the second sound accentuated. He had a slight cold and a good deal of soreness in lower part of his chest, probably due to coughing. He had been having slight nose bleeds. His blood pressure was 190, and his pulse 96. On August 6, at 5.30 A.M., he had such a severe nose bleed that I had to pack the nostril. His blood pressure immediately after this hemorrhage was 160. A little later it went up again to 180. While at Nauheim he was given 25 warm, termal, or brine baths. The blood pressure was gradually reduced to 160.

On his arrival the urine showed a trace of albumin, hyaline and granular casts. The second examination, August 11, 1909, showed a slight increase in albumin, but no granular casts. From this time on, until his departure, albumin and casts were gradually reduced, neither being present when he left Nauheim on September 10, 1909. The heart diminished in size until the left border extended only to the midclavicular line.

I saw this patient again in the spring of 1910. He was feeling decidedly better. There was a very faint trace of albumin in the urine, but no casts were found. He was taking iodide of potassium

on his arrival, and this was continued throughout the treatment, but no other medicines were given.

CASE V.—M. B., aged sixty years. For some years he had been having attacks of dizziness and headaches. He had typhoid fever in 1895, and a bad attack of gout in 1903. A marked eczema had existed for the last twenty years. He used alcohol to excess.

On his arrival in Nauheim, April 28, 1910, the blood pressure was 230 and the pulse 100. While here the patient was given thirty-six termal or plain brine baths. The blood pressure was gradually reduced, until on departure it was 175, with a pulse of 76 to 80. I have had several letters from this patient during the last winter, and he tells me that he has had no fainting attacks and his headaches are not as frequent or severe as formerly.

The urine examination on arrival showed albumin 0.003 per cent., and a few hyaline casts. One month later the urine showed neither albumin nor casts.

CASE VI.—Female, aged sixty years, arrived in Nauheim June 17, 1909. She had always been well except for headaches and neuritis. Three years ago her left arm became very painful and she was unable to move it. This lasted for six months, then gradually began to improve, and since then she has had practically no trouble in this arm. In April, 1908, she had a similar but less severe attack in the right arm, which began to swell and pained her constantly. I treated her in the winter of 1908 and 1909 in America, by baking, etc., and the arm improved. She remained well after that except for an attack of tonsillitis. She was a poor sleeper, had attacks of indigestion, and her bowels were constipated. Repeated examinations made during my treatment of her in America showed faint traces of albumin in the urine.

Her blood pressure on arrival at Nauheim was 128, and her pulse was 72. Her urine still showed a faint trace of albumin. While in Nauheim she was given twenty-two warm, saline baths. After two weeks the urine showed no albumin, she slept well, and was much less nervous.

CASE VII.—Male, aged fifty-three years, arrived in Nauheim May 5, 1909. He used alcohol and tobacco to excess. He had typhoid fever twenty years ago; double pneumonia ten years ago; four years ago he had a light attack of rheumatic fever which kept him in bed for two weeks. For many years he had had attacks of "intercostal rheumatism," and was subject to frequent attacks of bronchitis. For more than a year the patient had been able to sleep only two or three hours a night. In the latter part of March, 1909, while sitting in a chair after dinner, he fell to the floor, was dazed for about a half hour, could not speak, but knew what was going on about him.

After he reached Nauheim a physical examination showed the following: The breathing was short and jerky. The heart was

dilated, the left border extended one inch outside of the midclavicular line, and the right border the same distance to the right of the right edge of the sternum. The first sound was weak, the second aortic accentuated. The blood pressure was 140, and the pulse 96. His doctor sent with him to me six urine examinations that had been made between December 3, 1908, and February 10, 1909, in all of which a trace of albumin was found.

While at Nauheim the patient was given six plain brine baths and twelve termal sprudel baths and four sprudel baths. He left Nauheim on June 8, feeling well, but returned on July 11 and took twelve more baths. Examinations of his urine while there showed traces of albumin. His blood pressure was gradually reduced to 115, and the heart dulness diminished until it was about normal.

During the following winter the patient was much more comfortable, and was able to attend to his businesses, which are varied, and require a great deal of mental effort. He returned again in 1910, at which time there was no albumin in his urine, the blood pressure was 115, and the pulse 74. During his third sojourn in Nauheim the patient was given ten termal baths, ten termal sprudel baths, and four sprudel baths. During this time there was no albumin found in his urine, nor had there been any up to April, 1911, when I last heard from him regarding his urinary condition. He was feeling well, so well in fact that he did not deem it necessary to return to Nauheim last year. He was able to attend to his duties, had had none of the former fainting attacks, nor the shallow breathing. A member of his family recently told me that the patient was looking exceptionally well, and that his immediate family noticed much improvement in him.

Case VIII.—A manufacturer, aged forty-nine years, married. He had always been well except that he was nervous from overwork and could not sleep. On November 1, 1908, he was thrown from an automobile, received a severe blow on the head and fractured two ribs; he was able to be up and about in two weeks. While travelling, on December 2, 1908, he had a slight stroke, was conscious, but unable to speak. By the end of that month he was able to walk with the aid of a stick. In January, 1909, he was able to go out for drives, and on January 21 was able to go to the seashore, where he remained until early in May, coming to Nauheim in August of that year.

Examination showed that his heart was enlarged, the left border extending about one and one-half inches outside of the midclavicular line; the sounds were apparently normal; his blood pressure was 210, and his pulse 80. While at Nauheim he was given twenty-three warm, termal, or brine baths. The blood pressure was gradually lowered, until it was 162, and his pulse was 64, and the left border of his heart extended to just outside the midclavicular line. On arrival his urine showed albumin and hyaline casts. The cast

disappeared after two weeks, the albumin persisted until the last examination made on September 1, 1909, when neither albumin nor casts were present.

When I saw this patient in January, 1910, he was feeling fairly well. At that time his urine still showed neither albumin nor casts.

The patient returned to Nauheim from America in August, 1910. On landing in Hamburg he had an attack of dizziness, which lasted for a minute or two. He was nervous over this, went to a hospital and rested for a week. On his arrival at Nauheim examination showed that the left border of his heart extended to about the midclavicular line. His blood pressure was 170, and his pulse 68. He was given twenty-six warm, termal baths, to which mother lye was added after the eighth bath. His blood pressure fell to 160, and his pulse averaged 66. On arrival his urine again showed a faint trace of albumin, but no casts. Two weeks later the albumin had entirely disappeared.

CASE IX.—Male, aged fifty-seven years. He had typhoid fever in 1868, and pneumonia in 1902. For thirty years he had been treated for stomach trouble. For the last few years he had had difficulty from "gas pressing on the heart," always relieved by belching, and frequent dizzy attacks, especially in a crowded house, theatre, etc. He had pain in the precordial area, and had been taking nitroglycerin from four to six times a day for two years.

On his arrival at Nauheim, June 17, 1910, I found the blood pressure 170, pulse 70, and very irregular, and the heart enlarged to the left as far as the midclavicular line. The patient was given twenty-eight warm, saline baths, with the result that there was a gradual reduction of blood pressure to 152. Although he took no nitroglycerin during the last two weeks of his stay, he had no pain. I saw the patient in New York early in April, 1911, and he told me that he had not taken nitroglycerin during the entire winter, and had had no pain whatever. I found his blood pressure to be 150 at that time.

Urine examination on his arrival at Nauheim showed 0.006 per cent. of albumin, but no casts. Four weeks later there was a very faint trace of albumin present, and a few hyaline casts were found.

CASE X.—Civil engineer, aged sixty-two years, arrived at Nauheim May 19, 1909. For the sixteen years previous to this he had worked in an office. His stomach had been troubling him for two or three years, and he slept poorly. He had a good deal of headache; his bowels were obstinately constipated, and he was extremely nervous.

Examination showed that the patient was very thin and emaciated; there was marked arteriosclerosis; a small amount of cardiac hypertrophy, and his blood pressure was 180 and his pulse 80. While at Nauheim he was given twenty termal or plain brine baths, and three termal sprudel baths; between meals and at bedtime he

drank one and one-half to two glasses of milk, treated with lactic acid bacilli. His blood pressure was gradually reduced to 155 and his pulse to 70.

I saw this patient about six months after his treatment. He had gained fifteen pounds in weight, and was feeling better in every way. He was able to again attend to his work, and had no headaches.

Urine examination on arrival at Nauheim showed a faint trace of albumin, and a few hyaline casts. Five weeks later, at the time of leaving, the urine showed a very faint trace of albumin and no casts.

From the foregoing cases, and many more that I have treated, in which a probable nephritis was present, I am fully convinced that the Nauheim waters, when used as termal or plain brine baths at about 95° F. (35° C.), are beneficial in cases of Bright's disease, and that in treating cases in which albumin and casts are present the waters must be heated to above the normal temperature of the springs, which for Spring No. 7 is 86.2° F. (29.9° C.); and Spring No. 14, 90° F. (32.2° C.). Spring No. 12 is the only one which could be used at its normal temperature, namely, 94° F. (34.4° C.). I have found that the waters, when supercharged with carbonic acid gas as they come from the springs, are not suitable in such cases. Such water would, therefore, have to lie in storage tanks, to be used as termal or brine baths, and in that case would have to be artificially heated, as some of the temperature would be lost while in storage.

Cases I, VII, and VIII are cases that have been at Nauheim for more than one treatment, and showed a definite improvement in the kidney conditions each time they returned. Their condition at the end of the second year, and in one case the third year, was as good or better than at the end of the first year's treatment. The improvement in these cases seems to be of a permanent character

Cases II and III are cases which showed a marked improvement while taking the cure—Case II especially, in whom the albumin was reduced from a large amount to a faint trace, and the casts, which were present in large numbers on his arrival, entirely disappeared. Case III had 0.52 per cent. of sugar on the first examination made. While under treatment there were three other examinations made, and in none of these later examinations was any sugar found. I do not know whether sugar had ever been found before, so I am unable to draw any deductions as to this condition.

The blood pressure in all of these and in other cases, was lower after the course of baths. In the cases in which the albumin and casts entirely disappear the reduction in the blood pressure is more marked than in the cases in which the albumin and casts only partially disappear. This would lead me to suspect that in such

cases the high blood pressure is largely caused by the kidney condition.

TABLE SHOWING THE BENEFICIAL EFFECTS OF HOT NAUHEIM BATHS ON ALBUMINURIA AND HIGH BLOOD PRESSURE.

	Date.	Sex.	Age.	C.c. of urine in 24 hrs.	Sp. gr.	Per-cent. of albu-min.	Casts.	Blood pres-sure.	Pulse.
Case I	June 20, 1909	Female	37	1500	1014	0.003	0	160	102
	July 30, 1909			1650	1015.5	Trace	0	152	86
	Aug. 12, 1909			1500	1025.	0	0	130	74
	May 22, 1910		38	1200	1018.	0	0	145	90
	June 6, 1910			1400	1018.5	0	0	140	84
	Aug. 23, 1910			2000	1014.5	0	0	130	74
	July 21, 1911		39	1500	1025.	0	0	135	76
	Aug. 3, 1911			1800	1021.	0	0	130	70
Case II	July 28, 1909	Male	58	1500	1019.	0.014	H. and G., few	160	104
	Aug. 6, 1909			1650	1015.	0.012	H., few	130	80
	Aug. 17, 1909			1650	1013.	0.003	H., very few	130	80
	Aug. 25, 1909			1600	1014.	Trace	0	130	82
Case III	July 23, 1910	Male	58	1900	1019.	0.031	H., many G., few	190	92
	Aug. 1, 1910			1400	1018.	0.009	H., few G., very few	180	72
	Aug. 9, 1910			1800	1014.	0.008	H., few	180	68
	Aug. 25, 1910			1950	1010.	0.006	H., very few	180	72
Case IV	Aug. 3, 1909	Male	69	2150	1021.	Trace	H., few G., few	190	96
	Aug. 7, 1909			1050 +?	1021.5	0.006	H., few	160	80
	Aug. 21, 1909			1950	1021.	Trace	H., very few	165	80
	Aug. 26, 1909			1500	1020.	Trace	H., very few	180	80
	Sept. 10, 1909			1530	1024.	0	0	180	80
Case V	May 6, 1910	Male	60	1800	1014.	0.003	H., few	230	100
	June 6, 1910			1950	1008.5	0	0	175	78
Case VI	June 18, 1909	Female	60	1700	1015.5	Trace	0	128	72
	July 4, 1909			1800	1015.5	0	0	130	72
Case VII	May 20, 1909	Male	53	1000	1023.	Trace	0	140	96
	July 13, 1909			1100	1023.	0	0	130	76
	July 14, 1910		54	1500	1021.5	0	0	115	80
	Aug. 3, 1910			1550	1021.	0	0	108	62
Case VIII	July 4, 1909	Male	49	1500	1024.	Trace	H., few	210	80
	Aug. 7, 1909			1650	1024.	Trace	0	170	76
	Aug. 25, 1909			1750	1021.	Trace	0	185	64
	Sept. 1, 1909			1800	1014.5	0	0	165	68
	Aug. 8, 1910		50	1550	1025.	Trace	0	170	64
	Aug. 13, 1910			1800	1014.	Trace	0	160	66
	Aug. 25, 1910			2150	1009.	0	0	162	64
	Sept. 11, 1910			1900	1014.	0	0	160	64
Case IX	June 19, 1910	Male	57	1650	1017.	0.006	0	170	70
	July 13, 1910			1680	1023.	Trace	H., few	152	68
Case X	May 21, 1909	Male	62	1010	1022.	Trace	H., few	180	80
	June 23, 1909			2150	1013.	Faint trace	0	160	20

Just how these baths affect the kidneys is not at this time thoroughly understood. They do cause a relaxation of the entire muscular tissues, as evidenced by the fact that after taking from four to five baths patients are unable to do more than a small percentage of the walking or other exertion of which they were capable before beginning the baths. The baths also relax the per-

ipheral circulation, and this effect may, and possibly does, extend to all tissues of the body. Nauheim baths also cause marked elimination, especially through the skin and kidneys. I have often seen patients who came to Nauheim with a skin almost as dry as a sheet of parchment, after two, three, or four weeks of bathing have an active skin. Indeed, many of these patients remark that they have not perspired for years, even on exertion, but that now, after taking the cure, they perspire easily. This increased elimination through the skin doubtless is no inconsiderable factor in lessening albuminuria and lowering blood pressure.

REVIEWS

BIOLOGICAL ASPECTS OF HUMAN PROBLEMS. By CHRISTIAN A. HERTER, Professor of Pharmacology, Columbia University; Member of Board of Directors (and Physician to the Hospital) of the Rockefeller Institute. Pp. 344. New York: The Macmillan Company, 1911.

THIS book, edited by Susan Dows Herter, from a connected draft not in final form for publication at the time of Dr. Herter's death, represents the interesting and successful attempt of this well-known investigator in, and benefactor of, scientific medicine to set down, for his children, "his interpretation of biological laws in their bearing on human life, in the hope that they may prove of some service to persons who have faith that an understanding of such laws is frequently a help to intelligent and humane conduct."

The biological conception presented is that "human beings are no exception to the rule that all organized beings are in the process of adaptation to their surroundings;" "that all things living are in a state of seething flux, tending to construction or destruction." These tendencies are governed, as the author demonstrates, by quite definite biological laws, which conscious beings may understand and utilize, in order to bring about an intelligent adaptation of the individual to his group of conditions. This rationalistic conception is naturally opposed to the view that religious teachings are the best guide to conduct, and also to the view that each individual has to work out his own salvation by personal experience.

The argument is presented in four "Books" under the general headings: I. The Animal Body as a Mechanism. II. The Self-preservative Instinct. III. The Sex Instinct. IV. The Fundamental Instincts in Their Relation to Human Development. Under the first heading are presented the mechanistic conception of the human body, the problems of growth, reproduction, and inheritance, and a discussion of consciousness and the will. Book II, in four chapters, discusses the instinct of survival, the defences of the body, self-preservation, and the mental life, death, and immortality. Sex instinct is discussed in three chapters devoted respectively to sex and the individual, sex and social relations, and the male and female mind. Book IV presents arts and religion, education and the future of the race, and the fruits of education.

The treatment of these various phases of the subject is a mingling of scientific accuracy and scientific imagination, tinctured by a hopeful idealism of personal and social service which renders the perusal not only pleasing, but stimulating, and suggestive as well of possible concrete social effort. It is shown that the mechanistic view of human life does not lead to the hopeless resignation frequently considered as a necessary outcome of scientific fatalism, but, on the contrary, points the lesson of the great possibilities of developing personality through improved education, the increased opportunities for self-development, friendly surroundings, and mutual social service. In the discussion of "Death and Immortality" the idea is brought out that the cutting off of the prospect of heavenly rewards and hellish punishments "would lead to a greater considerateness in all human relations;" that instead of an obtrusive instinct of self-preservation based on a belief in personal immortality, it would develop a desire for mutual helpfulness, with, if necessary, self-sacrificial efforts, in the behalf, for example, of one's offspring. In the discussion of sex is emphasized the great educational and cultured influence of a higher regard (or affection) between the sexes and of the influence of this on ideals and general conduct of life. The refinement and control of the sex impulse, one of the most significant evidences of advancing civilization, the author associates, apparently in causal relation, with what Western nations regard as an improvement in the status of women. The chapter on "Education and the Future of the Race," with the discussions of the relative importance of "observation, memory, and the welding power of reason" as educational objects, and of education in its relation to the sexually based emotions present much of novelty and much that is of distinct value to parents, educators, and physicians; for that matter, this statement applies to the book as a whole.

The reviewer cannot refrain, though perhaps here he goes out of his way so to do, from an appreciation of Dr. Herter, in connection with this, his last message. The book before us exemplifies his character as a "student of man and of nature," and reminds us of his broad humanity and his devotion to the best ideals of his profession. In his work as physician, neurologist, chemist, and pharmacologist (and in the latter capacity he did much to develop experimental pharmacology in the medical schools of New York City), as a director of the Rockefeller Institute and physician to its hospital, as a consulting expert of the United States Department of Agriculture, as an investigator in his private laboratory, as founder and benefactor of the *Journal of Biological Chemistry*, and, with Mrs. Herter, of lectureships known by his name at the New York University and Johns Hopkins University, he was ever actuated by the ideal of service to American medicine and of the wider recognition of science in medicine. Investigator, bene-

factor, organizer, and advisor in all that pertained to medical service in this country, "a presence to be felt and known," he leaves with us, in the posthumous work here reviewed, ideals of effort and ideals of service, so tinged by a vivid scientific imagination, as to constitute a message destined to profoundly influence the progress of science in the profession he labored so earnestly to aid.

R. M. P.

A HANDBOOK OF PRACTICAL TREATMENT. In three volumes. By 82 Eminent Specialists. Edited by JOHN H. MUSSER, M.D., Professor of Clinical Medicine, University of Pennsylvania; and A. O. J. KELLY, M.D., late Assistant Professor of Medicine, University of Pennsylvania. Vol. II, pp. 865, 103 illustrations; Vol. III, pp. 1905, 47 illustrations. Philadelphia and London: W. B. Saunders Company, 1912.

IN the September (1911) issue of the AMERICAN JOURNAL OF THE MEDICAL SCIENCES (p. 444) appeared a review of Volume I of this System which is completed in Volumes II and III now before us.

The chief aim of this work is to present, in a reasonably small space, a record of the opinions held and the methods employed, in the treatment of disease, by men of recognized authority. These authorities have been chosen from all branches of medicine—thus medicine, surgery, and the specialties, including dentistry, are represented among the eighty-two contributors. Though contained in the space of three volumes, of about 950 pages each, the work may be considered an encyclopedia of the methods of treatment tested and accepted by scientific and authoritative practitioners engaged in the clinical care of the sick. The work is essentially clinical in character, yet the discussions in most instances, deal sufficiently with laboratory investigations to enable the reader to understand the rationale of the measures recommended.

It would be hypercritical for one to compare, in these volumes, the space devoted to therapeutic considerations. Any thought on treatment demands accurate and thorough diagnostic reasoning, and a work which judiciously correlates these will discourage the therapeutic romancer and so be a distinct influence for good.

It is gratifying to note the absence of long discussion, upon drugs and therapeutic measures whose uses are unknown and unsupported by experiment. One finds but few prescriptions and these are, for the most part, composed of drugs whose physiological action is known.

Not only is the treatment considered, but the characteristics of the various diseases are set forth in a manner unusually thorough for such a work; the practitioner, therefore, in considering his treatment thinks in terms of diagnosis, pathology, and prognosis.

So many false prophets have crept into public recognition by means of irrational, extravagant, or fraudulent claims for methods and cures that the union of the diagnostic and therapeutic reasoning of men of recognized standing in medicine constitutes the most important feature of this system.

Systems written, as they are, by authors of widely divergent style and experience are bound to present many variations in quality. The monograph will always be superior to the chapter written for such a work, yet one cannot deny the usefulness to the practitioner of such systems. For example, typhoid fever in Musser and Kelly's work is thoroughly discussed by the physician, the surgeon, the oculist, and the aurist, all of whom frequently consult in the care of this many-sided disease. A feature of the medical consideration of this disease is the detailed account of the steps in "tubbing" with admirable illustrations. The diet in typhoid is fully and practically dealt with. The use of vaccines and sera in typhoid is thoroughly yet conservatively considered.

Sir Clifford Allbutt writes of the diseases of the cardiovascular system. The chapter on the surgery of the heart, while brief, has perhaps the distinction of being the first upon this subject to appear in a work on treatment.

Tuberculosis is most exhaustively dealt with, being allotted 119 pages in a volume of 815 pages of text. The illustrations of institutions established for and of the apparatus employed in the fight against this disease are numerous and instructive. The writer has done well to set forth, in this comparatively small space, so clearly and thoroughly the enormous amount of work done upon the treatment of tuberculosis. This article demonstrates well the therapeutic triumphs resulting in the coöperation of the different branches of scientific medicine.

In the chapter on pneumonia the writer states that "the designation pneumococcic infection is preferable to croupous or lobar pneumonia" because "the inflammatory lesions in the lung may be the smallest part of the process going on in the body and because the extent of the lung involvement has not much to do with the severity of the illness. . . ." and on pages 271 and 272 is a most admirable exposition of the systemic character of the disease. While we have as yet no definite knowledge of how to attack pneumonia as a blood infection, it is to be regretted that the writer, who so clearly points to the systemic character of this disease, has not made a fuller reference to the work done in serum therapy, even though it were only to state where it has failed. Every year the practitioner realizes more keenly that the pneumococcus is not always the only organism at work in this infection, the *anatomical* diagnosis of which is "pneumonia," as the *anatomical* diagnosis "endocarditis" is often only the expression of a destructive streptococcus invasion. An emphasis of the failure of serum

therapy in the infection which we call pneumonia would go far to demonstrate the inefficiency of the drugs so commonly forced upon these unfortunate victims, and to train our minds to think that in antibodies, whether from the system itself or artificially administered, lies the secret of cure. There is, however, throughout the article an emphatic "hands off" to the meddlesome therapeutist.

Yellow Fever is the title of an article by the late James Carrol published posthumously. One reads with much interest the note appended to this chapter in which Dr. Juan Guiteras (to whom the article was submitted for revision after Dr. Carrol's death) states that "the disease is dying out; in a few years it will be a paleontologic study" (p. 571).

There is a section devoted to tropical diseases (not including yellow fever, cholera, etc.). It might perhaps have been wiser to omit this section. The diseases under this heading are rather superficially dealt with (except malaria), and on this account the book would hardly be useful to those engaged in the present-day energetic study of these maladies, both in the tropics, and at special laboratories. On the other hand, one would not attempt to treat an isolated case upon the meagre information supplied in this work. The bulk of the volume might with advantage have been reduced by omitting this section.

The first 103 pages of Volume III may be considered a clinical monograph upon dietetics in disease. In these pages are included diabetes, obesity, inanition, scurvy, rickets, osteomalacia, arthritis deformans, and gout. Throughout, only one stereotype prescription appears, and drugs are but rarely mentioned. The one prescription is the time-honored "Tr. Nux and Gentian," which has so long been a substitute for a square meal to the starving out-patient that perhaps it should be included among these dietetic considerations.

The feature of the article on diabetes is the diet tables. These differ from those usually appearing in works on treatment in that they are arranged with the aim of supplying the physician a dietary *for the varying phases* of the disease and the different metabolic needs of each case. It is well to add, however, that the text preceding these tables must be read in order to intelligently apply them.

It is refreshing to see but one page allowed to uric acid diathesis and lithemia, and this is devoted to ruling the term and the theory out of medical consideration.

The same fate falls to chronic articular rheumatism, which is disposed of in three-quarters of a page.

Under diseases of the digestive system the physician and surgeon are almost equally represented, and we find an effort throughout to encourage their coöperation early in the disease without necessarily the purpose of operating. The dentist, in whose field we

have come to recognize the source of some of the most destructive and sometimes fatal general septic infections, contributes an article upon general hygiene of the mouth and disorders of the teeth and gums. Too much emphasis cannot be placed upon the importance of including this subject in a work on general medicine. This article deals with those subtle diseases of the teeth and gums often overlooked by astute consultants and clinicians. The writer might perhaps have insisted upon this fact more forcibly and pointed out some of the joint diseases and other septic manifestations far removed from the real and curable cause.

The treatment of nervous diseases and diseases of the mind will be turned to with much interest. This section, taken with the section on hydrotherapy and mechanotherapy which appeared in the first volume will be found to offer much practical assistance to the general practitioner. I am not competent to speak for the alienist and neurologist.

Lack of space prevents further detailed consideration. The general practitioner, and in many instances the specialist, will find in these three volumes a reliable guide, and future authors of systems on treatment will do well to employ this as a model. The publishers have made attractive volumes, which in spite of their size and number of pages, are surprisingly light, and lie open well at almost any portion of the book. C. N. B. C.

Applied Anatomy. By Gwilym G. Davis, Associate Professor of Applied Anatomy, University of Pennsylvania. With 630 illustrations, mostly from original dissections and many in color by Erwin F. Faber. Philadelphia and London: J. B. Lippincott Co., 1911.

The object of this work is to show the relation of structure to function, whether it is normal function or function disturbed or impaired by injury or disease. The anatomy of the human body is studied in order that medical and surgical treatment may be intelligently applied, and consequently a book of this character possesses greater value than a mere treatise on anatomy, unless the latter is regarded simply as a storehouse of facts. There is a good deal of exaggeration of the value of a minute knowledge of anatomy to the surgeon, but there is no gainsaying the fact that in certain operations, such as tendon and nerve transplantation, plastic operations on joints, dissections of the neck for tuberculous nodes, hernia operations, and some others, the operator will do better work if his anatomical knowledge is exact.

This book seems to accomplish the task admirably, and includes a variety of anatomical and surgical facts, balanced and associated

so as to make an intelligent, interesting, and stimulating presentation of applied anatomy. There are, of course, omissions and statements to which others might take exception, but such must be unavoidable in the preparation of a work of this size by one writer. The purely anatomical portions seem more satisfactory to the surgeon than do the surgical portions, perhaps to the anatomist the reverse might be true, as it is disappointing, when referring to the applied anatomy of a certain operation or disease, to find the technical steps in the operation discussed briefly and without direct connection to the anatomy of the region. For instance, the extension in cancer of the lip is insufficiently treated, and in excision of the tongue no description is given of the anatomical points involved in removal through the neck; the description of excision of the rectum includes removal by the perineal or sacral routes, but no mention is made of the anatomical points involved in removal through the abdomen or by the so-called combined route, a matter of extreme importance. In the description of hernia the antiquated divisions of congenital inguinal hernia are well discussed, as are the various anatomical layers and structures entering into the formation of the canal and rings, but the relation of the sac to the cord, of the femoral vein to Poupart's ligament, etc., are not clearly given. The author seems at his best in the description of the joints, the chapters upon the shoulder and hip being especially praiseworthy; the abdomen, however, is not nearly so satisfactory.

The illustrations are clear, beautiful in execution, and have the faculty of showing what they are intended to show in the great majority of instances. A few, such as the one showing excision of the thyroid gland (Figure 184) convey no idea which might be useful. The general appearance of the book, its binding and printing, is excellent. G. P. M.

THE MORTALITY OF ALCOHOL. By EDWARD BUNNELL PHELPS, M.A., F.S.S. Pp. 75. New York: Thrift Publishing Co., 1911.

THIS is a statistical study of the causes of death in the United States by alcohol. The author is well qualified for such work, inasmuch as for over twenty years he has edited one of the leading insurance magazines in this country, and in other ways has maintained his interest in the cause of death. He gives comparative tables in which alcohol may have been the cause of death, either as a primary or secondary factor and compares his results with those of the United Kingdom. He comes to the conclusion that the mortality in which alcohol figures directly, indirectly, or even remotely is about 5 per cent., or about 6 in 100 deaths. This is somewhat lower than the estimate given by others, but, taking into consideration the difficulties which enter into such calculation, it is probably a just estimate. The work is well done, and is an interesting contribution. T. H. W.

INFECTIONS OF THE HAND. A GUIDE TO THE SURGICAL TREATMENT OF ACUTE AND CHRONIC SUPPURATIVE PROCESSES IN THE FINGERS, HAND, AND FOREARM. By ALLEN B. KANAVEL, M.D., Assistant Professor of Surgery, Northwestern University Medical School, Chicago. Pp. 447, with 133 illustrations. Philadelphia and New York: Lea & Febiger, 1912.

THE author has placed before the profession a work that has been much needed ever since surgery has been in existence. Fully appreciating the serious nature of hand infections, and realizing that in the past the treatment of such infections has been inadequate and often ill-advised, due to lack of anatomical knowledge, he has made a most complete study of the anatomy of the hand. By the use of numerous illustrations the relations of tendons, tendon sheaths, spaces, bursæ, and aponeuroses are shown. *X*-ray plates of bismuth injected specimens and serially sectioned anatomical specimens, show the shape, size, and weak points, by reason of which infection spreads from one fascial layer to another. The work is the most complete on this subject published today, being practically the only one that furnishes exhaustive anatomical descriptions with especial reference to their surgical application.

The book is useful alike to the general practitioner and the surgeon. The index and general arrangement is such that the diagnosis and treatment of a particular case is easily found and followed.

With such a mass of isolated and different facts all of more or less importance and yet so related as to demand mention together, it has been impossible to obtain a clear, concise, and adequate text, without repetition on the one hand, or too much cross reference on the other. The author has made the latter mistake, and consequently has made difficult reading, interrupted every few lines by a reference, which in its turn again refers the reader to a third place. This condition also exists in the legends under illustrations. The reader is referred to the legend of another illustration for the explanation of the lettering on the cut under observation at the time. In one or two instances the illustration and the text do not agree.

In several places the style is distinctly stiff, halting, and confusing to the extent of ambiguity. Too much space is consumed by introductory classifications and case histories.

With the exception of the few above-mentioned adverse criticisms the work deserves the greatest of credit.

The chapters on diagnosis and treatment are ably written and justly emphasize the fallacy of several of the older treatments. Packing with dry gauze for drainage is most justly and severely criticized. Gutta-percha is rightly given first place as a drain. Free incisions, by the bloodless method, are advocated.

In the postoperative treatment the writer advises hot antiseptic dressings, frequently changed, and partial Bier constriction for the first ten to eighteen hours.

A short clear *resume* is given of the literature on infections of the hand as well as the opinions of numerous surgeons on their methods of treatment.

A work fathered by ideas which give such wonderful results as are claimed by Dr. Kanavel is most certainly indispensable to every surgeon and general practitioner.

After reading these results, we better appreciate how little we knew of the surgery of the hand, and what poor results we obtained.

E. L. E.

THE SURGICAL CLINICS OF JOHN B. MURPHY, M.D., AT MERCY HOSPITAL, CHICAGO. Vol. I, No. 1, February 1, 1912. Pp. 133; 8 illustrations. Philadelphia and London: W. B. Saunders Company, 1912.

DR. MURPHY is recognized as one of the foremost teachers of surgery in this country; he is also an original thinker, and, what is more, has the faculty of setting his audience thinking. The series of volumes to be published bi-monthly, of which the first is now before us for review, is announced as a *verbatim* report, by an expert medical stenographer, of the remarks made by Dr. Murphy while operating, together with his discussion of such matters as are raised by the operation or the patient's disease.

The range of subject matter is wide, embracing surgery of mammary carcinoma, gastric ulcer, peripheral nerves, pleura and lung, joints, pelvic tumors, hemangioma, fistula in ano, etc. To the spectators of the operations under discussion his remarks during their performance no doubt conveyed some idea of what was being done. To others, who have not seen the operations discussed, no clear idea of the technique of the operation as applied to the individual case is afforded by interjections, exclamations, and ejaculations such as any surgeon utters after he has plunged *in medias res*. If anyone likes gibberish let him read the account of the operations called nerve anastomosis, commencing at page 25. The ulnar and median nerves are as hopelessly tangled in description as they seem to have been in cicatricial tissue, and we have been unable to come to any conclusion as to what really was the nature of the operation employed. "*Valde desirans, Pater optime, vires deficiunt.*" But the clinical lessons which Dr. Murphy derives from the abundant surgical material at his command, when stripped of too frequent pseudoscientific jargon, are clearly and intelligibly enunciated, and are of much value to the larger audience which these volumes will reach.

Might it not be well, in addition to the services of an expert medical stenographer, for the publishers to employ those of an expert medical proof-reader? Dr. Murphy evidently has had nothing to do with the "making" of the book, nor is he to have, so far as can be gathered from the publishers' announcements; and he will be quite as much surprised as anyone to see recurrent references to Henle, when Handley evidently is intended, to learn that in opening the abdomen the rectus fibers were misplaced, instead of displaced, etc. A. P. C. A.

Outlines of Psychiatry. By William A. White, M.D., Supt. Government Hospital for the Insane; Professor of Nervous and Mental Diseases, Georgetown University, Washington, D. C. Second edition; pp. 232. New York: Journal of Nervous and Mental Disease Publishing Co.

This is the second edition of White's *Outlines of Psychiatry*. It differs from the first in the fact that the whole subject of paronia has been rewritten and minor changes made. It is an excellent work of its kind for students and physicians who are not specialists in mental disease. T. H. W.

Practical Cystoscopy and the Diagnosis of Surgical Diseases of the Kidneys and Urinary Bladder. By Paul M. Pilcher, A.M., M.D., Consulting Surgeon to the Eastern Long Island Hospital; Late Surgeon to the German, Seney, and Samaritan Hospitals of Brooklyn, New York. 233 illustrations, 29 of them being in colors. Philadelphia and London: W. B. Saunders Company, 1911.

The introductory chapter deals with the indications for the use of the cystoscope. The first part is devoted to a discussion of cystoscopic technique, including the description of various European and American cystoscopes with their advantages and disadvantages, the care of instruments, and detailed instructions concerning the procedures of a cystoscopic examination. The author describes the appearances presented by a normal bladder, together with the technique of ureteral catheterization with both the direct and indirect cystoscopes. Fifty-nine pages are devoted to the discussion of diseases of the bladder as seen through the cystoscope, and the text is liberally illustrated with pictures of varying merit. Of the numerous functional kidney tests, the author prefers indigo-carmine, phenolsulphonephthalein, and

phloridzin, and he is of the opinion that these are only valuable in connection with the other and older methods of diagnosis, by the aid of ureteral catheterism. Diseases of the kidney are taken up from the standpoints of symptomatology, diagnosis, and indications for treatment, together with the cystoscopic findings commonly observed in each lesion. The therapeutic uses of the cystoscope are admirably summarized in the concluding chapter. Throughout the book bears the stamp of a man who commands a thorough, practical knowledge of his subject, to whom experience has been the guide in presenting the essential and omitting the unnecessary.

F. E. K.

DIE PALPABLEN GEBILDE DES NORMALEN MENSCHLICHEN KÖRPERS UND DEREN METHODISCHE PALPATION. By DR. TOBY COHN. Part 3, pp. 585; 33 illustrations. Berlin: S. Karger, 1911.

THIS is the third division of this work, the previous two being upon methods of palpation of the upper and lower extremities respectively. It is an excellent presentation, for it details the methods of palpation of all the structures of the head and neck, and as such should be of great value to all clinicians. T. H. W.

ONE HUNDRED SURGICAL PROBLEMS. THE EXPERIENCES OF DAILY PRACTICE DISSECTED AND EXPLAINED. By JAMES G. MUMFORD, M.D., Visiting Surgeon to the Massachusetts General Hospital; Instructor in Surgery, Harvard Medical School, etc. Pp. 354; 12 illustrations. Boston: W. M. Leonard, 1911.

THIS volume, which forms one of the "Case History Series," constitutes, as Mumford notes in his preface, a return to an older method of instruction—that by example instead of by precept. Writers used to collect several "centuries" of cases, each series consisting of one hundred case histories; and these were published in rather indigestible form. To be of any practical value these ponderous folios and quartos, written when Latin still was the universal language of science, had to be accompanied by an almost equally ponderous index; and to consult any series of similar cases in the tomes of Bonetus, Fabricius ab Acquapendente, Morgagni, or other similar worthies, requires a long working day, much of which is spent in studying the indices rather than the text. Dr. Mumford presents his case histories in palatable form, well arranged according to the regions or organs of the body affected, and accompanied with a modest but useful index, as well as a table of diagnoses.

The case histories are interesting, at times entertaining; and always convey important lessons of diagnosis, prognosis, or therapeusis, which are enhanced by the intercalation in the text of epexegetical commentaries and *obiter dicta* quite characteristic of the author. On page eleven, the misprint (?) gastrocolic omentum should, we presume, be changed to transverse mesocolon; for whereas a form of *anterior retrocolic* gastro-enterostomy was in fashion at one time in Germany, it does not seem likely that Dr. Mumford really advocates a *posterior antecolic anastomosis*, which is what is actually described. But when a surgeon drains every simple case of acute appendicitis for forty-eight hours, there is scarcely any intra-abdominal feat which he might be regarded as incapable of performing.

To say that the book is a very good one, is perhaps too faint praise; to say that it is not as good as some other somewhat similar and now classical volumes, such as Billroth's *Clinical Surgery*, for example, is within the truth. A. P. C. A.

PRACTICAL NURSING. FOR MALE NURSES IN THE R.A.M.C. AND OTHER FORCES. By MAJOR E. M. HASSARD, R.A.M.C., and A. R. HASSARD. London: Oxford University Press.

ALTHOUGH the authors have written this book primarily for the use of male nurses and orderlies, nevertheless a careful perusal of the work would be of advantage to the average practitioner, usually so negligent of the details of intelligent and careful nursing. The book would also make a good text-book for the hospital training school, as it is clearly written, compact, and thorough. The advice given is serviceable and practical; the faults are those of omission rather than of commission. J. H. M., JR.

PSYCHIATRISCHE VORTRÄGE FÜR AERZTE, ERZEIHER UND ELTERN. By PROF. DR. G. ANTON, Direkton der Klinik für Geistes- und Nervenkranke in Halle, a. S. Pp. 77. Berlin: S. Karger, 1911.

THIS small volume is designed for physicians, educators, and parents, and is of a popular variety. It takes up the question of normal and abnormal development, alcoholism, and epilepsy.

T. H. W.

PROGRESS

OF

MEDICAL SCIENCE

MEDICINE

UNDER THE CHARGE OF

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The Relation of Tonsillitis to Milk Supply.—C. E. A. WINSLOW (*Boston Med. and Surg. Jour.*, 1911, clxv, 899). A sudden increase in the cases of acute tonsillitis in certain parts of Boston and its suburbs, was talked of at a medical meeting in Boston on Thursday, May 11, 1911. By Sunday, May 14, there was an increase of 100 per cent. in the cultures examined by the laboratories of the health departments as compared with the previous Sunday. The disease differed clearly from ordinary tonsillitis. The most striking feature was the secondary enlargement of the glands of the neck, which in many cases followed the first sharp throat attack, and which in some instances was followed by a general invasion of the deeper tissues leading to sepsis, rheumatism, erysipelas, nephritis, and other maladies. Its severity increased with age, being usually mild in children. Most of the fatalities occurred in patients over fifty-five years of age. It occurred in a sharply marked epidemic culminating on May 12, 13, and 14. The epidemic, with the single exception of a clinically similar one among school children in Westchester County, New York, in February and March, was confined to Massachusetts. There seems to have been no connection between the two localities. Eighty-five to 90 per cent. of the patients had used milk from the same source, which amounted to less than 1 per cent. of the total Boston supply. Moreover, for the two months preceding the Boston outbreak, the disease was epidemic in the outlying towns around the suspected farm. Presumably a "carrier" infected the milk, producing the epidemic. At the farm itself the cattle are inspected four times a year by a veterinarian, and diseased cows forbidden; the sanitary conditions are regularly examined;

bacterial analyses of the milk are made six times a month; stringent rules are in force against human sickness, and milk from any farm where disease occurs is not used. Yet it cannot be doubted that contamination must, in some way, have occurred. However, against such outbreaks there is one certain safeguard, proper pasteurization with proper protection of milk from secondary contamination. To Winslow's knowledge no milk epidemic of similar throat disease has been hitherto reported in this country. In Great Britain, the phenomenon has been a common one. "Septic sore throat," so-called by the English, must be added to the list of dangers surrounding a raw milk supply.

Disturbances of Respiration Due to Nuclear and Intranuclear Disease.—C. F. Hoover (*Jour. Amer. Med. Assoc.*, 1911, lvii, 1734) states that the physiology of respiration involves a number of problems; the lung as an aerating organ; the mass movement of the blood; the partial pressure of carbon dioxide and oxygen in the alveolar air and circulating blood; products of metabolism; and finally the nervous control of respiration which may be modified by alterations in the bulbar respiratory centre. He cites loss of consciousness or apnea following morphine in incipient tabes; or the varying effects of that drug in cardiovascular disease with relation to Cheyne-Stokes breathing and slumber apnea. He concludes that the use of respiratory depressants must be employed with great caution when there is any reason for suspecting anatomic degeneration in the respiratory centre. Disturbed respiration also may originate from disturbed peripheral innervation of the lung, due to a neuromuscular reaction with spasm of the bronchioles and deficient lung ventilation, characterized by dyspnea of an emphysematous type, or with tachypnea without cyanosis or impared lung ventilation. The former is probably due to vagus neuritis, and as an example Hoover mentions a case of mediastinal tuberculosis. The latter is probably due to excitation of the pulmonary branch of the vagus. As examples, Hoover mentions the experiments of Baglioni, who observed that excitation of the pulmonary branch of the vagus produced marked tachypnea and more prompt arrest of the heart with lesser stimulus than when the central end of the vagus was excited. This was born out clinically in Hoover's experience by 2 cases of aneurysm, when the tachypnea was immediately relieved by atropine.

Sahli's Hemoglobinometer.—A. E. Boycott (*Proc. Roy. Soc. Med.*, London, 1911, Path. Sec, p. 43) reports that the two simple hemoglobinometers with a standard composed of hemoglobin or some derivative, are the modifications of the Gowers instrument introduced by Haldane and Sahli respectively. In the former the permanent standard is made by sealing up CO hemoglobin in an atmosphere of coal gas; in the latter it consists of acid hematin. The samples have to be converted before the comparison be made. Though the latter is the simpler instrument, it suffers from material inaccuracies. The standard does not seem adjusted to any definite strength of hemoglobin, normal blood varying from 80 to 120 per cent. with different instruments. In comparing a series of readings with two Gowers-Haldane and Sahli instrument, Boycott observed that, on the whole, the Sahli gave 17

per cent. too low, with the error varying between 8 and 24 per cent. The same sample of oxalated blood from an experimental animal was measured five times in parallel with the two instruments. The largest difference with the first was 5 per cent.; with the Sahli, 22 per cent. Thus Boycott concludes that the Sahli is not an instrument of precision, probably because the pigment is not in solution but in suspension, and because one cannot rely on always making the mixture in precisely the same way. For when blood is mixed with acid, the size of the particles of which the colored precipitate is composed, presumably varies with the precise method of mixture. Hence the depth of tint seen by transmitted light is not always the same, even when the same quantity of the same blood is mixed with the same quantity of dilute acid. (The remarkable variations in Sahli instruments have seemed to the reviewer so great as to render them unsafe for ordinary clinical work.—W. S. T.)

A Characterisitc (?) Feature of the Cardiogram.—C. PEZZI (*Compt. Rend. Soc. de Biol.*, Paris, 1911, cxxi, 646) reports that among the numerous signs of adhesive mediastinopericarditis some suggest the presence of concomitant endopericarditis. Pezzi has studied 3 cases in which this condition might legitimately be suspected. In 2 the diagnosis of mediastinopericarditis was confirmed by radioscopy. The cardiogram showed one common feature, lack of any auricular wave. This is explained by assuming that the presystolic wave is due to pressure exerted by the blood driven through the auricle on the apical portion of the ventricle. This, if surrounded by adhesions, becomes nondistendable. Thus no auricular contraction is recorded on the cardiogram. Pezzi considers that this may be an important sign of endopericardial adhesions with apical localization. It must be observed on a number of cases. Finally to be of value, the venus pulse tracing must show an auricular systole. It is this contrast which makes the sign of value.

Compensation by Displacement of the Thoracic Viscera in Pulmonary Tuberculosis.—CHARLES LESTER LEONARD (*Med. Record*, 1912, lxxxi, 1) reports that any alteration in the capacity of the lungs must be compensated for, or the excursion of the walls surrounding must be decreased. Marked alterations in the position of the intrathoracic viscera occur in the earlier stages of tuberculosis without visibly changing the excursion of the thoracic walls, to compensate for the destruction by the disease of the normal expansive tissue. Such can be demonstrated by the stereoröntgenogram. Displacements are of four kinds—displacement of lung by superficial pneumothorax; of heart upward and into anteroposterior position; of heart and aorta to to left or right. The stereoröntgenogram adds to the knowledge obtained by other methods of physical diagnosis by detecting superficial areas of pneumothorax, by showing that the aorta is generally displaced with the heart, and by making evident the effect of fibroid change in displacing the viscera through contraction. Finally, owing to its rapidity, the stereoscopic röntgenogram is more accurate than the fluoroscope, though it does not show the movements of the diaphragm.

Fatty Compounds as a Factor in the Etiology of Appendicitis.—Williams believes that appendiceal concretions are made up of compounds of saturated fatty acids, and that excess in secretion or defect in absorption of calcium and fatty acids may be their origin. A large increase of calcium soaps is found in the submoca and mucosa of some appendices, in certain cases producing marked thickening. Thus, by cutting off the nutritive supply, bacterial invasion may be made easy. ANTHONY (*Jour. Med. Research.*, 1911, xxv, 359) has studied 48 cases, using various fat stains to differentiate fatty acids and calcium soaps. Her conclusions are that while neutral fats and slight amounts of fatty acids occur in control cases, in acute and chronic appendicitis fatty acids and calcium soaps predominate, and seem to increase with the age of the patient, especially between the ages of twenty-five and fifty years. Thus in general, her observations confirm Williams' views, which are furthered by irregular cultural findings.

The Importance of Estimation of the Urea in the Blood in Bright's Disease.—FERNAND WIDAL (*Bull. et mêm. Soc. méd. d. hôp. de Paris*, 1911, 3 s., xxxii, 627). Recent communications have emphasized the importance of the amount of urea in the blood for the prognosis of Bright's disease. Its determination may furnish the only element of certainty obtainable. The normal amount of urea varies between 0.15 to 0.50 grams per liter of blood serum. If more than this, there is nitrogenous retention, which the organism cannot tolerate for any length of time. For example, patients whose blood contains 1 to 2 grams per liter of urea rarely live more than a year; 2 to 3 grams, months or weeks; more than 3 grams, a very short time. In cases of renal obstruction, however, a grave prognosis should not be given, as after the obstacle is once removed the kidney may resume its regular function, and nitrogenous retention cease immediately. In pregnancy an analagous mechanism is assumed by Weill and Wilhelm in regard to nitrogen retention, which they have seen rapidly disappear after delivery. Possibly the same kind of phenomenon occurs in certain acute nephritides in which renal functions may be markedly but temporarily involved. But in chronic Bright's disease retention is more permanent. Certain clinical symptoms suggest nitrogen retention; loss of appetite alone or accompanied by gastro-intestinal troubles; stupidity, often the prelude of terminal coma; finally, of great importance, albuminuric retinitis. But there may be no relation between symptoms and the condition. On the whole, the estimation of the urea in the blood of patients ill with Bright's disease furnishes exact evidence for prognosis, and makes it possible to determine at times a degree of illness which would not be suggested by anything in the patient's general condition.

On the Anti-pneumococcal Powers of the Blood in Pneumonia.—H. E. EGGERS (*Jour. Infect. Dis.*, 1912, x, 48) has undertaken to determine whether or not in cases of lobar pneumonia, an increase of antibodies, and more especially of opsonins, could be demonstrated by the plate method. In a number of cases he preserved serum and leukocytes until an entire series was obtained. Mixtures were then made of penumonia serum, pneumonia leukocytes, normal serum, and normal

leukocytes, which were incubated on blood agar plates with pneumococci of varying virulence isolated from pneumonia sputum. The amounts used were the same. After varying lengths of time the number of colonies were counted. From his observations, Eggers concludes that in lobar pneumonia there is development of antipneumococcal bodies, usually progressing up to the crisis; reaching its maximum at or a little after this time, and lasting for variable periods. That opsonins play a part, at least, in the process is indicated by the fact that the number of plate colonies was in some degree dependent on phagocytosis; that bodies not concerned in phagocytosis, presumably agglutinins, are present is suggested by the fact that the colonies were decidedly fewer in the mixture containing pneumonia serum, than in that containing normal. Finally cases in which the increase of antipneumococcal power did not occur, presented irregularities either in course or termination.

SURGERY

UNDER THE CHARGE OF

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The Relations of the Flexures of the Colon to the Kidneys and Kidney Tumors.—HAUSMANN (*Zentralbl. f. Chir.*, 1911, xxxviii, 1657) says that it is a generally recognized fact that kidney tumors are characterized by the demonstrable location of the large intestine in front of them. It is known also that occasionally a tumor not of the kidney may develop between the layers of the mesocolon behind the colon, like a tumor of the kindey. This rule governs tumors of the kidney because the flexures of the colon are adherent to the anterior surfaces of the kidneys, that of the left flexure higher up than the right, the latter being attached over the lower pole. Hausmann has shown that by deep and gliding palpation, the hepatic flexure can be determined to occupy an abnormal position. The sinking of the right flexure is usually, but not always, associated with nephroptosis. If the latter condition is only slight, the flexure can be felt below the kidney. If the nephroptosis is marked, the kidney must first be pushed upward before the flexure becomes palpable. During the ptosis the flexure lies either behind or above the kidney. In 80 autopsies there was no attachment of the right flexure to the kidney in 26, the flexure being always ptosed and movable, sometimes very movable. In these the position of the

flexure was varied and changeable, sometimes above, sometimes in front of, and sometimes behind the kidney. The right flexure can be palpated only when it is attached to the kidney or is only lowered. In the one case it is felt in front of, and in the other below the kidney. When it is behind the kidney, the ascending colon can be followed by palpation upward to the kidney, and the transverse colon from the left to the kidney. When the kidney is pushed upward the covered portion becomes palpable. Tumors of the normally situated kidney lie behind the flexure of the colon. Tumors of a ptosed kidney can lie behind the flexure of the colon, when the flexure has not lost its attachment to the kidney, or it may not have the colon in front of it when the flexure has lost its attachment to the kidney. In the latter case, the tumor lies in front of the colon. Such a relation is less likely in the tumors of the left kidney than in those of the right, because the flexure reaches higher and has a more firm fixation to the kidney on the left side. By gliding movements of the finger tips, deeply sunk, the various portions of the large bowel and stomach are accessible to palpation, and can thus be located. The small intestines are not accessible to palpation, except the end of the ileum before it joins the cecum. Hausmann desires that surgeons as well as internists should avail themselves of this method of diagnosis, and particularly that they should prove at the operation the facts elicited by palpation before the operation.

Thrombosis of the Superior Mesenteric Artery Simulating an Abdominal Tumor.—Thévenot and Rey (*Arch. gén. d. Chir.*, 1911, v, 1234) report a case of this kind because it presented aspects which differed from those of the typical forms. A man, aged sixty-seven years, was admitted to the hospital, June 12, 1910, for urinary disturbances due to an enlarged prostate. About nine months later he had a suppurative orchitis, and six months later still a perineal abscess which was incised. About a month later, during the dressing of the wound, the patient was suddenly seized with a violent pain in the right flank of the abdomen, and soon developed symptoms of shock. The pain was located below the false ribs, and radiated to the sternum and lower limbs. There was no nausea or vomiting, no stool since the night before, and no tympanities. At the site of greatest pain, a movable mass was palpated, the size of two closed hands, with a notch on its upper surface. It occupied nearly the whole space between the false ribs and the anterior superior iliac spine, and did not move with respiration. Its dulness was continuous with that of the liver. The lumbar region was painful on pressure, and lumbo-abdominal pressure elicited slight ballottement. There was no fluid in the abdomen and nothing was learned by rectal palpation. The temperature was 37°, pulse, 120, and rather feeble. On the following day the peritoneal facies and the grave general condition continued, and the extremities were cold. The other symptoms were the same, and on the third day the patient died. At autopsy there was found a mass of intestinal loops, violet black in color and distended, corresponding in position to the mass felt in the abdomen before the opening was made. The notch in the mass was formed by a loop of intestine. After separating the coils of intestines, it was found that the lesion was located about 2 meters from the pylorus, the intestine was markedly distended and discolored

for a distance of 50 cm. The adjacent mesentery was profoundly changed. For a breadth of three fingers, it was thickened, indurated, and edematous, of a brownish black color, and the surface was covered with small elevations. On opening the intestine there escaped a considerable quantity of pitchy, blackish liquid. After washing the surface of the mucosa there could be seen ecchymoses, vivid red spots, and some ulcerations, but no perforations. The rest of the intestine was normal. On dissecting the mesenteric vessels, the arteries being largely sclerotic, there was found in one of the branches of the superior mesenteric artery a grayish clot adherent to its walls. On the distal side of the clot the arteries were empty but the veins were engorged with blood. The typical symptoms of an obliteration of the mesenteric arteries are as follows: Early vomiting of the peritoneal type, which is sudden and severe in origin, and may contain blood; intestinal hemorrhages, sometime abundant; diarrhea, especially free at the beginning of the affection, later becoming sanguinolent; general meteorism; and subnormal temperature. The sudden development of a tumor in the abdomen, co-existing with a sudden violent pain is especially significant.

Surgical Treatment of Acute Pancreatitis—Körte (*Ann. Surg.*, 1911, lv, 23) says that the diagnosis of pancreatitis is very uncertain, and can never be made absolutely, owing to our not having as yet found any pathognomonic sign. The signs of peritoneal inflammation are the most prominent feature. When operating for peritonitis, in looking for the point of origin, the pancreas should be considered, and particularly when the inflammation is confined to the upper abdomen. The finding of an odorless, serosanguinous exudate, and even of greater importance, the discovery of small flecks of fat necrosis, is direct evidence of pancreatitis. Early operation is of the greatest importance. Of 16 cases operated on during the first week, 11 recovered, while 5 died. Of 14 operated on in the third and fourth week, one-half died. It is to be hoped that, as with peritonitis, the cases will be sent earlier to the surgeon. As certain results are not to be expected from early operation in this condition as in appendicitis and chlecystitis. The best results were obtained in those cases in which operation was undertaken before irreparable changes had occurred in the gland, in those which did not go on to necrosis, and also in those with encapsulated, inflammatory, hemorrhagic effusions in the neighborhood of the inflamed gland. Good results were also obtained in those cases of localized pus pockets in the pancreas, or in those in which there was a very limited necrosis, as in the acute hemorrhagic inflammations. It is fair to infer that only a small number of cases of acute pancreatitis reach the stage of necrosis, the majority dying before that stage is reached. We must strive to operate before gangrene of larger portions of the gland takes place, to drain the secretion, and where it is possible, to limit the advance of necrosis. This is more difficult in some cases because of the rapid necrosis of the inflamed and hemorrhagic, infiltrated gland. In fact, three or four days after the beginning of the illness it is possible in a few cases to find the gland already advanced in necrosis. The median incision in the early stage is by all means the best, as we can thus reach and treat the diseased organ, and also the gall passages best. The peritoneal exudate should be immediately sponged or washed out with hot saline solution. The pancreas should

in every case be exposed, best through the gastrocolic ligament or from the right side by the liberation and retraction of the duodenum. The approach through the transverse mesocolon is seldom to be advised. The important thing in all operations to consider is that all unnecessary manipulation of the inflamed parts should be minimized, as the danger of collapse is otherwise markedly increased. The exposed organ is, after breaking through the peritoneal covering, incised with a blunt instrument and drained with tubes and gauze strips. The gall passages are also at the same time to be freely exposed for inflammation or empyema. The lumbar incision should be reserved for those particualr cases in which there is retroperitoneal collection of pus, and also in the few cases which are operated on in the later stages. Pus in the subphrenic space is best reached through an epigastric incision, and in questionable cases can be sufficiently drained through a transpleural operation. In the stage of abscess formation, hemorrhage from pancreatic and neighboring bloodvessels is a very positive danger, and one which is not entirely obviated through the early operation. Thrombosis of the larger venous channels also occurs, with its consequent septic sequelæ. The pancreatic fistula which develop from the occurrence of tissue necrosis in acute pancreatitis have the tendency to heal spontaneously. The Pawlow-Wohlgemuth diet cure should be employed in each of these cases.

Absorption in Ileus and Peritonitis.—Enderlen and Hotz (*Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1911, 755) say that while we are well able to make the clinical diagnosis and construct at the bedside a satisfactory conception of the anatomical picture of the disease, in striking contrast is our lack of knowledge of the cause of the development of the clinical picture, the local anatomical and functional disturbances in the intestine. They undertook to determine by experimentation the nature of the absorption in the small intestine in the various stages of ileus and peritonitis. The experiments were carried out on dogs and in coils of intestine situated at various levels. They tested the absorption of sodium chloride and sugar solutions in different coils, in different concentration, and under varying pressures. In one group of experiments the absorption of the normal intestine was studied, in a second group the absorption in peritonitis, in a third in ileus. A localized peritonitis either does not disturb at all or only in slight degree the absorptive capacity of the intestine involved. On the contrary, we may assume that the local inflammation in consequence of the increased blood supply or inflammatory hyperemia, increases the absorption. This was shown in the experiments by the increased absorption in those dogs in which this function was already depressed. In the early stages of peritonitis the absorption is not decreased. It is first seriously disturbed with the development of general intoxication. In the final stage of peritonitis it is generally much depressed. It can be increased by an excessive secretion. It is believed that the toxic or bacterial general sepsis influences the activity of the intestine by a vasomotor disturbance. In ileus the absorption in the obstructed portion of intestine is much decreased. In all the experiments it was seen that the afferent empty coils of intestine had absorbed a greater quantity of fluid contents than the obstructed segment. This shows that the portion of intestine involved by the ileus has a diminished

absorptive capacity. With the employment of a 5 per cent. saline solution, there was found a marked fluid secretion in the lumen of the intestine. The quantity of this excess of secretion in the ileus coils was twice as large as in the afferent coils, and it was found further that this excess of secretion after seventeen hours was still undiminished. The irritation of the concentrated saline solution has also a much more intensive effect than in the normal intestine. To neutralize the high concentration an enormous quantity of fluid and mucus is produced. The absorptive capacity in the whole intestine, even in that portion not involved in the obstruction is diminished. In a localized peritonitis absorption in the afferent intestine is good, but with the development of general peritonitis absorption in the whole intestine gradually diminishes until stagnation and finally an excess of secretion occurs. There is no question concerning the necessity of early operation in ileus or peritonitis. Unnecessary manipulations within the abdomen are to be avoided. Under certain circumstances one must be satisfied with an enterostomy without seeking to ascertain the nature of the obstruction. The value and effect of the enterostomy is generally recognized. Various factors which damage the intestine are overcome. After the disappearance of the distention there is an inhibition of the irritation and of the disturbances of the blood circulation. Peristalsis and absorption in the intestinal tract are restored, provided that the gut has not reached an advanced stage of paralysis. The exhausted intestine, tired out by the unsuccessful efforts of the increased peristalsis, will not be able to carry off the fecal masses. There will be little necessity of stimulating this by physostigmin, strychnin, and other peristaltic stimulants. Opium should not be administered, but the bowel left to itself.

A Study of Sprain-fracture.—Ross and Stewart (*Ann. Surg.*, 1911, lv, 71), because of the importance of sprain-fracture as attested by its frequency and location (near joints), made a careful study of this condition from a clinical and experimental point of view. Clinically, the history of sufficient injury with a small, sharply localized area of swelling and acute tenderness over a region of tendonous or ligamentous attachment is diagnostic of sprain-fracture, and should be considered as such whether or not *x*-ray verifies the diagnosis. Tenderness of lesser degree may surround an acutely tender area. Tenderness lasts, as a rule, from five to twenty days, longer in improperly treated cases; however, those few cases with symptoms as given above, in which tenderness disappears within forty eight-hours, are, nevertheless, sprain-fractures. The acute tenderness referred to is unmistakable; when the spot is firmly pressed on the patient invariably winces. The following conclusions are reached: History of sufficient injury, with a sharply localized area of swelling and acute tenderness over a region of ligamentous or tendonous attachment, means sprain-fracture. *X*-ray is not essential for the recognition of sprain-fracture. The external malleolus is probably the most frequent seat of sprain-fracture. About 15 per cent. of all fractures are sprain-fractures. That condition, formerly called severe sprain, is sprain-fracture. The condition resulting from stretching of soft parts is better termed strain. Sprain-fracture is probably a part of the pathology of every dislocation. If in doubt as to whether or not sprain-fracture has occurred, treat as a sprain-fracture.

THERAPEUTICS

UNDER THE CHARGE OF

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The Action of Atophan in Normal and Gouty Subjects.—DEUTSCH (*Münch. med. Woch.*, 1911, lviii, 2652) believes that atophan is by far the best remedy thus far proposed for the treatment of acute gout. It is not surprising that its action is not so marked in chronic gout with marked anatomical changes in the joints. It is also impossible to say as yet whether kidney or heart complications so often associated with gout will be influenced by its use. He summarizes the action of atophan as determined by many different observers, and mentions the different theories advanced for its action. In healthy individuals the administration of small doses of atophan is followed by an increase of the uric acid excretion that is manifest within an hour after the drug has been given. Larger doses of atophan, up to 5 grams, caused an increased uric acid excretion that persists during the following day. The increase in the uric acid ranges from 30 to 300 per cent., and occurs in individuals on a mixed diet and those on a purin-free diet. The purin basis, phosphoric acid, total nitrogen, and total sulphur are not changed, nor is the total amount of the urine increased as a rule. After discontinuing the remedy the uric acid remains at a slightly lower level for one or two days, but soon returns to its former level. With the prolonged administration of atophan the amount of uric acid excreted remains at a higher level when compared to the amount excreted before the drug was given. Similar results are obtained in gouty subjects by the administration of atophan. In addition Frank and Bauch have found that gouty individuals are able to excrete uric acid introduced intravenously, or nucleinic acid given by mouth if they receive atophan at the same time. Deutsch gives the details of a number of carefully conducted experiments that in general confirm the above-given facts. He does not draw any definite conclusions from his results. Deutsch used atophan in the treatment of 25 cases of gout with good results. In some of the patients gastro-intestinal symptoms, such as abdominal pain, regurgitation of sour fluid and diarrhea, occurred as a result of the use of atophan. These did not occur when the remedy was combined with sodium bicarbonate.

The Treatment of Intestinal Amebiasis.—MUSGRAVE (*Jour. Amer. Med. Assoc.*, 1912, lviii, 13) says that in acute or chronic amebic dysentery characterized by frequent stools with blood and mucus, and usually accompanied by tenesmus, ipecac given in large doses is a most valuable drug. In the majority of instances its administration is followed by a rapid subsidence of the symptoms, and quick convalescence. In a smaller percentage of such cases it apparently does no

good whatever, and in a not insignificant number its administration is followed by an aggravation of the symptoms which in some grave cases may at least hasten an unfavorable termination, particularly if nausea and vomiting results from the treatment. Musgrave denotes these cases as clinical dysentery, "but besides these typical cases there are many cases without well-defined symptoms, but with etiological and pathological basis for the diagnosis of amebic dysentery. The latter group of patients do not react so well to the ipecac treatment. Musgrave advises giving colon irrigations with patients in the knee-chest position once or twice a day. Two or three liters may be given in this way, and at times patients are able to retain as much as four liters. Thymol, quinine, and silver nitrate solutions are the three that have proved to be of the greatest value. Of these Musgrave prefers thymol and he recommends the following formula because of the insolubility of thymol in water. Thymol, alcohol, āā 25; glycerin, q. s. ad 250. Add 10 c.c. of this solution to each liter of water used in the irrigation. Quinine solution is made up in the strength of 1 to 1000 or 1 to 500 of quinine bisulphate in water. Quinine solutions frequently give rise to constitutional effects produced by its absorption from the bowels, particularly in patients who are able to retain enemas for considerable periods of time. Nitrate of silver, in solutions of from 0.1 to 1 per cent. in distilled water, frequently proves of decided benefit. In his conclusions Musgrave says that each case must be a law unto itself as to the choice of the methods of treatment. The irrigation treatment is the main reliance, particularly in what may be termed average or usual cases. Ipecac is particularly valuable in the presence of clinical dysentery and may be tried either alone or in combination with bowel irrigation. The substance used in enemas should be arbitrarily changed from time to time in cases in which satisfactory progress is not being made by any one method of treatment.

The Combination of Salt-free Diet with Bromide and Iodide Therapy.—Survonat and Cremieu (*Rev. de Méd.*, 1911, xxx, 762), in their conclusions, say that a chlorine-free diet is a valuable aid to bromide therapy. This fact is explained by the substitution of bromine for chlorine in the body tissues. They suggest that a similar substitution may take place when iodides are given with a chlorine-free diet. Survonat and Cremieu also warn that toxic symptoms may be produced by small doses of either bromide or iodide when given in this way.

The Treatment of Late Diphtheria Complications with Intraspinal Injections of Antitoxin.—Bingel (*Deut. Arch. f. klin Med.*, 1911, civ, 374) says that in the convalescence of diphtheria we are often confronted with grave symptoms that, according to him, are the result of the action of the diphtheria toxin. These symptoms are sudden vomiting, delirium alternating with more or less apathy, and more or less evidence of circulatory failure, such as marked pallor and weakening of the pulse. Treatment must be prompt and energetic to combat this poisoning, and Bingel recommends intraspinal injections of diphtheria antitoxin after withdrawal of a corresponding amount of spinal fluid. The author has been impressed with the lack of benefit

of the ordinary procedures to combat the symptoms of cardiac failure occurring after diphtheria, and reports good results by this method of treatment. He suggests that the intraspinal injection of diphtheria antitoxin be made before the development of these grave symptoms, as a prophylactic measure. When the poison has produced marked changes in the myocardium or brain no benefit can be hoped for from these injections. This treatment relieves promptly when there are no such changes, and shows its value by rapid relief of vomiting and retching, and improvement of the circulation. No untoward effects were observed as a result of the injections, except a slight rise of temperature following the injection. The details are given of 11 cases treated by this method, of whom 8 recovered.

Salvarsan Therapy in Scurvy.—TSUCHINSKY and IWASCHENZOW (*Münch. med. Woch.*, 1911, lviii, 2671) have tested the action of salvarsan in scurvy because of the fact that the mouth symptoms are largely due to infection by mouth spirochetes. Repeated small doses of the drug caused a notable reduction in the number of spirochetes with corresponding improvement of the mouth symptoms. However, they do not attribute any especial curative action of salvarsan upon the disease scurvy.

The Prevention of Fever after Injections of Salvarsan.—WECHSELMANN (*Münch. med. Woch.*, 1911, lvii, 1510) thinks that the rise of temperature and constitutional symptoms following intravenous injections of salvarsan are due to dead bacilli in the distilled water used in making the solution. Consequently he has adopted the method of distilling and sterilizing the water for the saline used in the solution of the salvarsan immediately before the treatment. He says that with this technique he has given more than 150 injections in his office without any fever occurring within four hours after treatment. He considers salvarsan less toxic than mercury.

Vaccine Inoculation, Prophylactic and Curative, of Typhoid Fever.—PHALEN (*Jour. Amer. Med. Assoc.*, 1912, lviii, 9) gives in brief the record of prophylactic typhoid inoculation in the British, German, and American armies. He says that nowhere do we get such convincing evidence for the practice of typhoid immunization as from the records of our own army. Up to the present time over 60,000 men have completed the inoculations, and among this entire number and covering a period of nearly three years, but 12 cases of typhoid have developed, and no death has occurred. The record of the Maneuver Division in camp at San Antonio, Texas, during the last summer has been most instructive. An army division, having an average strength of 12,800 men, all inoculated, occupied the same camp from March to July, and in this command but one case of typhoid developed. This was a mild case in a hospital corps man who had not completed the inoculations necessary for protection. During the same four months there were 49 cases of typhoid in San Antonio, with 19 deaths. Between three and four thousand men were in camp at Galveston during this same period, and in this command no case of typhoid occurred, while the city of Galveston furnished 192 cases of typhoid during the existence of the

camp. The city and the camp had the same water, milk, and food supply, the only difference being that the camp had been protected by inoculation. About 3000 men were scattered along the Mexican border, mostly in small camps, many of them in localities where typhoid was present, yet of this command only one man contracted typhoid, which ended in recovery. Phalen ascribes this practical immunity to typhoid fever entirely to the inoculations. He says that treatment of typhoid fever by inoculations is still in the experimental stage. The fact that the dosage given and recommended by different writers varies between one million and one billion bacilli, shows that this practice is very unsettled. The total number of cases treated by typhoid vaccines is small, but they have been carefully observed by competent observers working with small groups of patients. Phalen thinks that the sum of opinions of these men is unquestionably favorable. Inoculation shortens the fever period, and the total duration of the disease, and it also markedly reduces complications and relapses. Very pronounced amelioration of headache, gastro-intestinal symptoms, and toxemia is usual. All the observers agree that even in the cases in which it causes no improvement it has done no harm. The dosage employed in the earlier cases usually ran from 10,000,000 to 100,000,000. With increasing confidence the dosage has been increasing, and now it is usually from 100,000,000 to 500,000,000. It is probable, according to Phelan, that with increasing dosage, even better results than thus far reported will be obtained. The mortality in all the cases collected from the literature by Phalen was 4.9 per cent., and he believes that the vaccine treatment may reduce typhoid mortality below its heretofore irreducible minimum.

PEDIATRICS

UNDER THE CHARGE OF

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Sodium Benzoate in Artificially-fed Infants.—CLIFFORD GRULEE and WALTER BUHLIG (*Archives of Pediatrics*, 1911, xxviii, 849) determined the general action of sodium benzoate on the bacteria in milk, and studied the effect of the drug in artificial milk mixtures. They used one part of milk as a control, one part contained sodium benzoate 0.1 per cent., and one part contained 1 grain of benzoate to every 6 ounces of milk. The milks were plated every twenty-four hours, and the bacteria counted per cubic centimeter. From these experiments they deduced that with 1 grain of the benzote to 6 ounces of milk (0.04 per cent.), or in 0.1 per cent. strength, the number of bacteria in milk does not differ materially from the number in control milks; that sodium benzoate in the above strengths has a slight inhibitory effect on some

bacteria; that sodium benzoate is a fair preservative only when few living bacteria are present. The clinical observations consisted in administering sodium benzoate in one-half and one-grain doses in each feeding to infants on artificial mixtures. Some of the infants showed before and during the benzoate administration malnutrition, rickets, gastro-intestinal disorders, and tetany. Grulee and Buhlig give complete histories of 9 cases, including milk formulas, treatment, and results. They conclude from these observations that sodium benzoate to the amount of 2½ to 5 grains in twenty-four hours in artificially fed infants, ranging in age from a few weeks to almost two years, produces no recognizable symptoms, even though these children may be suffering from gastro-intestinal disturbances of a serious nature.

Salt-edema in Older Children.—Franz Hamburger (*Münch. med. Woch.*, 1912, lviii, 2500) calls attention to the fact that increase or decrease of sodium chloride in the diet has quite an influence on edema. In nephritis or lost cardiac compensation in the adult, edema can be produced and intensified by increasing the sodium chloride in the diet, and then diminished by a reduction of the same. Infants without heart or kidney lesions react similarly, especially where disturbances of nutrition are present. It is possible to accomplish this in older children with normal heart and kidneys. It is essential, however, that some abnormal condition be present to obtain the effects—usually some disturbance of general nutrition. Hamburger cites one case in full, that of a child aged five years, who had suffered a long time from malnutrition and was emaciated. The heart and kidneys were not diseased. Under an increased sodium chloride diet the child gained 3 kilograms in two months, but developed general edema in a marked degree, which disappeared rapidly when salt-free diet was exhibited. The gastric disturbances had disappeared during the process, and the heart and kidneys remained normal. Increase of sodium chloride in the diet will occasionally cause profuse diarrhea, and edema will not develop. Many children with malnutrition or cachexia will react to this test with the appearance of edema. The inference is strong that infants, children, and adults may be constitutionally impaired by ingesting too much salt, and develop edema although the kidneys remain intact.

The Diazo Reaction in Scarlet Fever and Serum Sickness.—S. S. Woody and John Kolmer (*Archives of Pediatrics*, 1912, xxix, 12) have made a study of the diazo reaction in scarlet fever, diphtheria, and serum sickness to determine its diagnostic value in cases of serum sickness exhibiting a scarlatiniform eruption. A large number of diphtheria cases develop serum sickness with rashes of a scarlatiniform, morbilliform, or urticarial nature. The first type offers the greatest diagnostic difficulty, as it develops from the second to the sixth day after administration of antitoxin. Kerr and Rivier found the diazo reaction invariably absent in scarlatinaform erythema due to serum disease, and the latter found it positive in 56 per cent. of cases of scarlet fever. Woody and Kolmer studied altogether 502 cases as follows: 375 cases of scarlet fever; 56 cases of diphtheria; and 37 cases

of serum sickness. The scarlet fever cases showed the diazo reaction positive in 8.53 per cent. The largest number of positive reactions occurred during the first week of the disease, when the rash was present, and gradually diminished and disappeared with convalescence. Uncomplicated cases of diphtheria showed a general average of 5.4 per cent. positive. Rashes in serum sickness gave a general average of 10.8 per cent. positive. Sixteen cases of the scarlatiniform type showed one positive diazo, or 6.2 per cent. Of 18 urticarial types 2 were positive, or 11.1 per cent. In 3 cases of the morbilliform type none were positive. In the one positive case of the scarlatiniform type, in which there were no other symptoms of scarlet fever, there ensued an uneventful recovery and no desquamation. The positive diazo here may have been due to the diphtheria. Woody and Kolmer place little value on the diazo reaction as a point of differential diagnosis, because it is positive in only 15 per cent. of scarlet fever cases during the first week, and because it was positive in 12.9 per cent. of diphtheria cases during the first week. The reaction was found positive in 75 per cent. of cases of measles. Therefore a negative reaction in a case of morbilliform eruption would be of value in differential diagnosis between serum sickness and measles.

The Hot Air Treatment of Infantile Eczema.—JENNY PERLMANN (*Münch. med. Woch.*, 1912, xlix, 85) reports the results in the treatment of eczema of infants by the application of hot air by means of a cabinet. The results were excellent, not only in the moist forms of the disease, but also in the dry forms, such as the papular, squamous, and vesicular. Most of the cases were infants who had been treated for some months without benefit, by means of the usual salves and ointments. They were considerably under weight, and were in constant distress from itching. After a few treatments the itching and restlessness were markedly improved. During the course of the treatment the children gained in weight, the temperature became regularly normal, and all but the inveterate cases healed in a remarkably short time. It was found that this treatment acted exceptionally well in cases of furunculosis. The furuncles dried up rapidly under the influence of the heat, and soon disappeared, and the further spread of the condition was stopped. A daily application of the hot air was given for from five to ten minutes. The eczematous areas were treated with olive oil between the hot air treatments. Perlmann treated 35 cases in the last six months. The time necessary to cure varied from seven days to three and four weeks. A strict regulation of diet was carried out in each case.

Simultaneous Occurrence of Acute Inflammation of the Heart in Children.—A. FORELL (*Münch. med. Woch.*, 1912, xlix, 142) calls attention to the query of Max Herz, who saw so many identical cases of acute endocarditis at the same time in Vienna, that it suggested to him the appearance of an infectious disease in the form of an epidemic. Forell reports a similar outbreak of acute heart disease in children, in which seven children living in a small area bounded by three city streets were taken acutely ill with identical symptoms. Five of the children were taken ill within a period of forty-eight hours, one child four days

before, and one five days later. They ranged in age from five to ten years. In every case the disease affected acutely and primarily the heart. In no case was there swelling of any of the joints and in but 1 case faint signs of a chorea. The patients comprised two pairs of sisters from two families living close together; one patient lived across the street from them, and the earliest case and the latest case in point of development of the disease lived on the same street as those already mentioned, in adjoining houses. In most of the cases the myocardium alone was involved, in 2 cases the myocardium and endocardium, and in 1 case, in addition to these, the pericardium. One case began with a frank catarrhal angina. In 2 other cases, a history of beginning sore throat was given. The onset in every case was acute, with high fever and marked constitutional prostration, and severe pain over the præcordium. The fever invariably dropped within two or three days, and the temperature remained subnormal for several days. In the myocardial cases arrhythmia and irregularity of the cardiac action developed only after the temperature fell. Only one case, an endocarditis, with myocarditis, showed this symptom before the fever fell, during the second day of the disease. The cases with endocarditis showed involvement of the valves only at the beginning of the second week. Cardiac dilatation occurred in all cases, an increase to the left being always the first sign. In all the cases a true mitral insufficiency existed. Some of the cases showed an enlarged liver and a number had severe nose-bleed. Cultures from the nose and throat in all the cases were negative for the diphtheria bacillus. A blood culture on one case during the febrile stage was negative. The causal factor is problematical. Probability would indicate an acute rheumatic infection from the slight choreic symptoms in one case, the typical rheumatic pericarditis in another, and, finally, the fair recovery of the patients.

The Treatment of Post-diphtheritic Paralysis by Antitoxin.—Max Crohn (*Münch. med. Woch.*, 1912, lix, 84) reports 3 cases of post-diphtheritic paralysis which responded to subcutaneous injections of diphtheria antitoxin. In all the cases strychnine, digitalis, arsenic, etc., had no effect on the progress of the paralysis. In the first case, a boy, aged twelve years, the paralysis began three weeks after the onset of diphtheria and affected the skeletal muscles in general and the soft palate in particular. Myocarditis and dyspnea developed also, and the paralysis was progressive until 2000 units of diphtheria antitoxin were given subcutaneously. The progress of the condition was halted, and within three days general improvement was marked and recovery was uncomplicated. In the second case a post-diphtheritic paralysis of the legs occurred in a boy, aged four years. One thousand units of antitotoxin caused marked improvement within four days. In the third case, post-diphtheritic paralysis occurred in a boy, aged twelve years, four weeks after the onset of the disease. The legs, back, and soft palate were first affected. There was no improvement under arsenic, etc., and in two weeks there was general muscular paralysis with bronchitis, fever, and marked involvement of the soft palate and throat. Three days after the injection of 2000 units of antitoxin the boy was able to drink fluids, and in eight days phonation and swallowing were intact, and there was a marked improvement in the general

muscular weakness. Kohts, who first reported results from this treatment, used large doses of antitoxin, 25,000 to 35,000 units. Crohn achieved as good results with small doses, and observed a prompt and steady amelioration of the condition after one injection. The antitoxin destroys any diphtheritic remnant in the body, whether bacilli or toxin, and makes impossible a continuation of the nerve destruction. There is no necessity for repeating the antitoxin if the first dose causes a halt in the progress of, or a slight improvement in the paralysis. There was no development of serum-sickness in these cases, owing possibly to the smallness, both of the initial and of the subsequent dose of the antitoxin.

OBSTETRICS

UNDER THE CHARGE OF

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Puerperal Mortality.—UNTERBERGER (*Arch. f. Gynäk.*, 1911, Band xcv, Heft 1) discusses in a lengthy paper the puerperal mortality in Mecklenberg-Schwerin from 1886 to 1909. In the twenty-four years he finds that 1 in 488 cases of labor terminates in death from puerperal sepsis; 1 in 1480 cases ends in death from tuberculosis; 1 in 1948 cases from death by eclampsia; one in 3454 cases died from embolism; 1 in 3759 from postpartum hemorrhage; 1 in 4291 from hemorrhage complicating placenta prævia; 1 in 8170 from nephritis; 1 in 10,892 from atony and failure of expulsive pains. These statistics are interesting as denoting the relative frequency of the different causes of death in communities where medical matters are under careful supervision.

The Treatment of Hereditary Syphilis.—BAISCH (*Monats. f. Geburts. u. Gynäk.*, 1911, Band xxxiv, Heft 3) believes that where clinical signs of syphilis are present in both parents, the indications for the treatment of both are manifest. Where a healthy mother gives birth to a macerated fetus, spirochetes will be found in the adrenals of the fetus and point to infection in the father at some indefinite period. In the Munich clinic, in 190 mothers having such an experience, paternal syphilis was proved in 146. Recent studies show that every mother giving birth to a syphilitic child is infected, no matter how apparently sound she may be. The infection travels by the spirochete through the placenta from mother to child. The possibility of a healthy mother giving birth to a syphilitic child is denied. In all cases, then, treatment must be addressed not only to the father, but to the mother as well. Where syphilitic children are born apparently healthy many show a positive Wassermann reaction. It may be that children are occasionally born of a syphilitic mother who cannot be demonstrated

to be syphilitic. Where the mother becomes infected toward the end of pregnancy the child may not share the infection. It is certainly true that every child born of suspicious parentage must be subjected to modern methods of examination, and if a positive result is obtained, appropriate treatment must be at once begun. Where the reaction is negative the child must be kept under observation and subjected to repeated and minute examinations. The old treatment of syphilis by mercury and potassium iodide was unsatisfactory, so far as the children were concerned, and in many cases after prolonged treatment a syphilitic mother gave birth to a syphilitic child. Salvarsan, in Baisch's experience, has been given intravenously in doses of 0.4 to 0.6 gram, the average being 0.5 gram. It is more active in its effect upon the spirochete than mercury, and seems to have a better and more rapid result. In 35 cases the Wassermann reaction was always positive, though several injections are needed in old cases to change the reaction. Apparently it is not necessary clinically to secure a positively negative reaction. Should healthy children be born, the treatment must be considered successful. It cannot be denied that occasionally mercurial treatment is successful, although in the majority of cases one cannot depend upon it. In 3 cases treated by salvarsan the results were favorable, so far as the child was concerned. Where salvarsan was injected into the mother Baisch saw no results upon the child. He believes that it is advantageous to treat the patient during the puerperal period, even though she is apparently well, if she gave birth to a macerated fetus. It is often impossible to give it in one injection, and the opportunity should be utilized in the puerperal state to secure this.

The Treatment of General Septic Peritonitis.—Morison (*Brit. Med. Jour.*, October 28, 1911) reports among others the case of a multipara in her eighth pregnancy, who introduced a bone crochet needle into her uterus with the object of procuring abortion. The needle broke, and but half of it was recovered by the patient. She walked into the hospital, and immediately after admission had a chill, with a pulse of 120 and a temperature of 106° F. The lower part of the abdomen was tender on pressure, and vaginal examination showed only a three months' pregnant uterus. Operation was done as soon as possible, and the cervix was first divided anteriorly to permit exploration. The needle was found lying across the interior of the uterus, and vaginal hysterectomy was performed. The peritoneum covering the uterus was acutely inflamed, with effusion. The patient recovered. The trend of this paper, and the discussion upon the subject, is to urge prompt operation upon all cases of septic peritonitis. Three depressing factors in these cases require attention: The first is exposure, and it is essential that these patients be kept warm during operation. The second is the danger of prolonged anesthesia, which can be reduced to a minimum by skill on the part of the anesthetizer by reducing the anesthesia to the shortest possible time, and having everything in readiness before the operation is undertaken. In Morison's experience, chloroform was used, in which the laryngeal reflex was deadened, and then ether was substituted. The third and most important factor was the avoidance of rough manipulations of the abdominal

contents. The general principles of drainage were carried out, and in severe cases the patient received about two pints of normal salt solution in a vein, with a small dose of adrenalin at a temperature of 100° F. Proctoclysis was employed, and in some patients as much as twenty-two pints was given in twenty-four hours. Lavage of the stomach was useful. The limited use of morphine proved valuable. Purgatives judiciously employed were also useful.

GYNECOLOGY

UNDER THE CHARGE OF

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Vesicocervical Fistula.—KÜSTNER (*Zeit. f. Geb. u. Gyn.*, 1911, lxix, 402) describes an operative method which, he says, in a large series of cases has never failed to cure this condition the first time. He makes a transverse incision in the anterior fornix, and then by blunt dissection, or with the aid of the scissors, completely separates the bladder from the cervix to a point at least 1 cm. above the upper edge of the bladder fistula, believing this to be an essential point in any operation for this condition. A few sutures of fine aluminum-bronze wire are now introduced in such a manner as to close the bladder opening, and to attach it, and a portion of the bladder wall below it, to the intact portion of the surface of the cervix *above* the cervix fistula. The latter is disregarded, or it may be transformed into a fissure by cutting through the bridge of tissue which separates it from the external os, this procedure sometimes being of value in facilitating orientation and application of the sutures. If the vagina is narrowed by scar tissue, or the uterus is very difficult to pull down, much help may be secured by making an extensive Schuchardt incision, or by completely circumcising the portio.

Combination of Tuberculosis and Carcinoma.—Two interesting cases of this condition affecting the female sexual organs have been very carefully studied by v. Franqué (*Zeit. f. Geb. u. Gyn.*, 1911, lxix, 409). One of these was a case of primary carcinoma of both tubes associated with tuberculosis, neither of these processes affecting the uterus or ovaries, except for the presence of carcinomatous metastases on the surface of the latter. In the second case the ovaries were free, both tubes tuberculous only, and the uterine mucosa tuberculous, with carcinomatous areas in the cervix. This patient had been operated on for tuberculous peritonitis several years previously. Franqué thinks that in both these cases the tuberculosis was the etiological factor in the development of the carcinoma, probably the inflammatory infiltration and disintegration of the connective tissue caused by the tuberculosis favoring the epithelial invasion, a condition which has come to be well recognized by most pathologists as occurring in other

organs of the body. It seems probable that in these cases the carcinoma was of multicentric origin, not only in the sense of arising simultaneously in different areas, but also because of arising from both superficial and glandular epithelium. Franqué calls attention to the atypical epithelial proliferation, somewhat suggestive of cancer, frequently seen in connection with tubal tuberculosis; he has found this in his case as well, but does not believe that this is a forerunner of true carcinoma, as the latter appears to him to arise from the unchanged epithelium. He believes that the tuberculous irritation is responsible for both this atypical, benign proliferation, and for the development of carcinoma, and sees in the demonstrated relationship between tuberculous and malignant conditions of the internal sexual organs a further reason for not treating the former conservatively unless operation is specially contraindicated.

End-results of Surgery in Neurasthenia.—Reynolds (*Boston Med. and Surg. Jour.*, 1912, clxvi, 275) says that he fully agrees with the conclusions reached at the joint meeting of the American Surgical and Gynecological Societies in 1910 with regard to this subject, namely, (1) that neurasthenia is fundamental in the individual, *i. e.*, that typical neurasthenias occur only in persons who are ill-developed in some particular, (2) that neurasthenia is never *in itself* an indication for surgical treatment, and (3) that it *is not necessarily a contraindication.* He believes that in the past many surgeons have neglected the second proposition, and have performed operations that were never indicated, thereby bringing surgery into much disrepute in this connection, but also that equally grave mistakes have been made by internists and neurologists, who have failed to realize the importance of the third proposition, thereby withholding treatment which might in many instances have been curative. The diagnosis in any given case as to whether the neurasthenia is really due to local lesions, or whether it is of general origin, but is referred by the patient to an etiologically unimportant symptom, is often of the greatest difficulty, but is also of the utmost importance. If after most careful examination it becomes evident that the nervous breakdown has preceded the development of local symptomatology, it will usually be safe to assume that the latter is not the responsible factor; operation should, therefore, be avoided in these cases, even in the presence of a demonstrable but non-lethal lesion. If, however, the local trouble appeared first, and especially if it has persisted unchanged for years, followed by the development of a neurasthenia, the former will generally be found to be the causative condition. A cardinal principle in treating local lesions of a gynecological nature in neurasthenics should be to avoid prolonged minor treatment, for even in cases where this is locally successful, it is almost certain to be generally deleterious. In gynecological work, moreover, radical extirpation of organs is usually accompanied by so much nervous shock as to be equally inadmissible in the treatment of neurasthenic patients; conservative operations should therefore be given the preference in all cases where they are at all possible. By a rational application of conservative surgery of the female reproductive organs in cases where a distinct lesion seemed to be at the bottom of the trouble, Reynolds has been able to cure 82 per

cent. of patients in whom neurasthenia figured prominently in the symptom-complex; he believes, therefore, that if these principles are carefully carried out many supposed *malades imaginaires* will be found susceptible of a prompt cure, or at least of being restored to a fair degree of health and happiness.

DERMATOLOGY

UNDER THE CHARGE OF

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Toxic Dermatoses.—HARTZELL (*Jour. Cutan. Dis.*, March, 1912), in a paper read before the American Dermatological Association, in a symposium on the toxic dermatoses, concludes that such diseases as erythema multiforma, urticaria, dermatitis herpetiformis, pemphigus, and some other bullous affections of uncertain classification, are toxemias due to a great variety of toxic substances which may be introduced into the body from without, may arise from auto-intoxication, or from local or general bacterial infection; and that, although they differ from one another sufficiently in their symptomatology and course to entitle them to be considered as separate and distinct affections, they are essentially one in their pathogenesis. Fordyce (*Ibid*), in a paper on "The Influence of Anaphylaxis in Toxic Dermatoses," read in this same symposium, regards the various forms of disease constituting the so-called erythema group of skin diseases as phenomena of anaphylaxis, and believes that this affords the most plausible explanation of the *modus operandi* of the many causes of these affections.

Syphilitic Erythema Nodosum.—JANSON (*Dermatologische Zeitschrift*, Band xviii, Heft 12) reports a case of secondary syphilis with universal small papular eruption, tendency to hemorrhages, and an erythema nodosum precisely like the idiopathic form of this affection. From the observation of his own case and a study of other cases in literature, Janson concludes that there can be no doubt about the occurrence of a specific luetic erythema nodosum. The treatment of this variety of the disease is that of syphilis, but iodine in combination with other antiluetic remedies seems to be especially effective.

LEVISEUR (*Jour. Cutan. Dis.*, November, 1911) thinks the resemblance between this form of erythema nodosum and Bazin's disease (erythema induratum) is becoming more and more apparent. He regards it as a malignant form of syphilis (precocious tertiary), which is frequently preceded, accompanied, or followed by gummata.

Telangiectases in Children, in Association with Wasting and Protracted Diarrhea.—Fearnsides (*British Journal of Dermatology*, February, 1912) reports a series of 6 cases, all in children, in which telangiectases, erythema, and purpura occurred in connection with wasting and protracted diarrhea. The erythema was present in all the cases and in 5 was associated with telangiectases. Purpura occurred in 2 along with the erythema and telangiectases. The erythema was found usually on the distal portions of the extremities, while the tëlangiectases were on the thighs and parts of the trunk exposed to pressure. Edema was present in all the cases. Fearnsides concludes that these various rashes were the expression of vascular dilatation, and were the direct result of the wasting brought about by the prolonged diarrhea.

Some Papulosquamous Syphilides and Their Relation to Psoriasis and Parapsoriasis.—Ravogli (*Archiv f. Dermatologie und Syphilis*, Band cxi, Heft 1), under the above title, discusses the not infrequent close resemblance existing between some papulosquamous syphilides and psoriasis, and reports briefly some cases in illustration. He concludes that in some instances psoriasis follows syphilis as a metamorphosis of the papular process. In some cases it is a hybrid form to be referred to an attenuated luetic taint; and although not the direct result of syphilis, it can, in many instances, be regarded as a parasyphilitic affection. (We cannot agree with some of Ravogli's conclusions as to the relationship between syphilis and psoriasis. There is no evidence to prove that the two diseases are even remotely related etiologically.—M. B. H.)

The Treatment of Darier's Disease by the Röntgen Ray.—Ritter (*Dermatologische Woch.*, Band liv, No. 6) reports a case of Darier's disease successfully treated by means of the Röntgen ray. In the beginning of the treatment small doses were employed (3 to 5 *x*), but without result. Larger doses (10 *x*) were then tried, and to these the affection reacted very promptly, the hyperkeratoses disappearing without leaving any scar. Gradually the greater part of the body was exposed to the rays, the result of the treatment being especially brilliant on the face, which was greatly disfigured by hyperkeratoses. The treatment lasted about eighteen months, and Ritter believes the good results permanent.

Eosinophile Cells and "Mastzellen" in Vesicular Eruptions of the Skin.—Pultz (*Arch. f. Dermatologie u. Syphilis*, Band cxi, Heft 1), who has studied a large number of cases of various vesicular eruptions such as eczema, scabies, herpes, etc., has found a considerable number of eosinophile cells and "mastzellen" in the vesicles of these diseases. In most scabies vesicles and in those of eczema eosinophiles and "mastzellen" were found together, the number of the former, however, greatly exceeding the latter. In the lesions of herpes labialis and progenitalis there was a high-grade eosinophilia and a marked increase in the number of "mastzellen," but in those of 2 cases of herpes zoster neither variety of cell was found. In the lesions produced by vesicants there was likewise a high-grade eosinophile and basophile leukocytosis.

PATHOLOGY AND BACTERIOLOGY

UNDER THE CHARGE OF

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A Partly Successful Therapy for Cancer.—A preliminary communication of great interest was recently made to the Medical Society of Berlin upon this subject by WASSERMANN, KEYSSER, and MICHAEL WASSERMANN. It forms the subject of an article in the *Deutsch. med. Woch.*, December 21, 1911, and of a leader in the *Brit. Med. Jour.*, January 6, 1912. These workers have been seeking a remedy that would exert a selective action upon neoplastic tissues leading to the destruction of those tissues without effect upon the normal body cells, and have met with a partial success in a compound of selenium, tellurium, and eosin. No particulars of its exact qualitative or quantitative nature are to hand, but it may be said that the use of a large dose has led to the dwindling and sometimes to the disappearance of carcinomatous growths in laboratory animals. The growths experimented upon were in some cases transplantation, and in others spontaneous tumors; the curious statement appears that if the required dose be injected into a healthy animal the entire skin of the animal becomes pink (from the eosin), whereas, if a newgrowth be present this does not occur, but the tumor becomes an intense red, indicating a special affinity between the drug and the tumor cells. Tumors as large as a cherry have disappeared under the treatment.

Inflammatory and Arteriosclerotic Kidney Disease.—J. F. GASKELL (*Jour. of Path. and Bact.*, January, 1912), working at Freiburg, contributes a paper containing the results of a painstaking examination of more than 60 kidneys. He concludes that the glomerular and arterial changes give a means of differentiating certain forms of disease that is accurately in keeping with the clinical history of the cases. Among inflammatory cases Gaskell segregates those in which the renal change accompanies ulcerative endocarditis, indicating them as embolic focal nephritides; in these a characteristic fibrinous exudate and leukocytic infiltration affecting some of the glomerular capillaries is found. In such cases, the renal affection is a secondary one and the patient dies from the disease of the heart, and not from uremia, as is likely to happen in the true glomerulotubular cases which are readily separated from the above by the microscopic changes. Permanent inflammatory changes, apart from those seen in ulcerative endocarditis, are always due to true glomerulotubular nephritis, the effects of which are to be seen in different stages, the acute, known generally as acute parenchymatous nephritis, the subacute commonly called "large white kidney," and the sclerotic, usually known as secondary contracted kidney, less well designated "small white kidney." In general, Gaskell

supports the views of Löhlein. The renal changes due to general vascular disease mark an entirely distinct group, which includes those due to senile arteriosclerosis and those in which a true primary contracted kidney, due to arteriosclerosis of the small arteries, are seen. In the former, the senile form, the arteriosclerosis is mainly seen in the aorta and other large vessels, whence it spreads to the renal arterioles, with marked atrophy and glomerular degeneration. Although a high degree of shrinkage may occur, death usually is due to intercurrent disease, and clinically the group is an unimportant one. The latter form, where there is seen a genuine contracted kidney, is seen in cases where a general, well-marked arteriosclerosis of small arteries and arterioles leads to death from such a cause as cerebral hemorrhage.

The Causative Agent of Scarlet Fever.—Much activity has lately prevailed in attempts to convey to monkeys the virus of scarlet fever; prominent among workers have been CANTACUZÈNE, BERNHARDT, VIPOND, LANDSTEINER, LEVADITI and PRASEK. Vipond holds for the causative agent a large, readily-cultivated bacillus. The three authors, last named, inoculated into the tonsils and under the skin, scarlatinal products and produced fairly satisfactorily the signs of the disease, but their animals showed blood cultures of streptococcus; the material does not produce symptoms unless inoculated into the tissues, and one is compelled to think that their experiments allow streptococci to complicate the results, although their own belief is that there are effects that can be attributed only to another virus. More definite, perhaps, are Bernhardt's results with material scraped from the tongues of patients, injected partly under the skin, partly in the mouth-tissues of an ape, of which the inguinal nods were then emulsified and injected. This was repeated, and the third ape, treated with emulsion that had been filtered through a Berkefeld filter, showed the exanthem, strawberry tongue, fever, subsequent desquamation, especially of the hands and feet, and occasional albuminuria. He thus thinks the disease to be due to a filterable virus, and appends a description of small bodies in the filtrate, which stain readily by Giemsa's method, and which he has found in the nodes and kidneys of individuals dead from the disease.

A Medium for Diphtheria Bacilli.—RANKIN (*Journal of Hygiene*, xi, 1911) has obtained very accurate results in the recognition of diphtheria bacilli by the use of a medium composed of sheep's blood serum, 3 parts; bouillon, 1 part; glucose, 0.5 per cent.; potassium sulphocyanide, 1 part; and 2 per cent. of a 5 per cent. solution of neutral red. The bacillus of diphtheria grows in rose-colored colonies, which are readily distinguished from the colonies of those few other microbes (chiefly cocci), the growth of which is not retarded by the medium.

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